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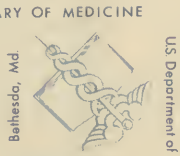
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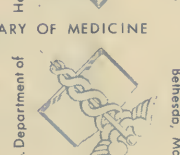
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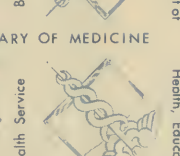
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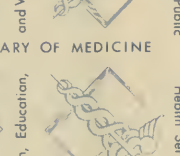
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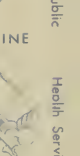
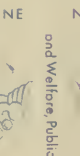
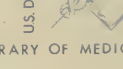
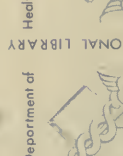
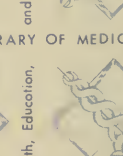
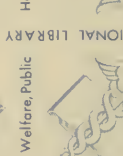
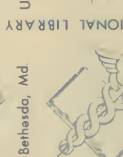
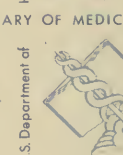
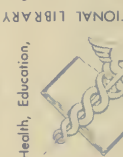
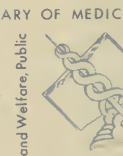
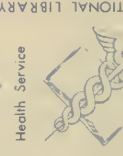
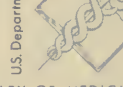
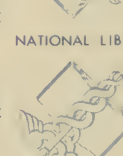
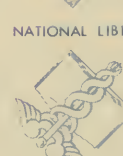
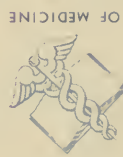
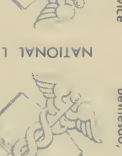
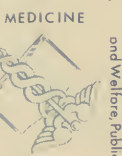
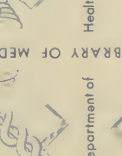
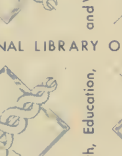
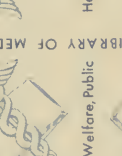
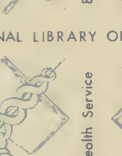
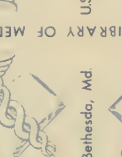
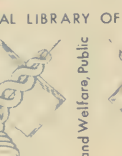
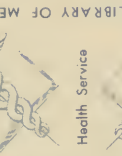
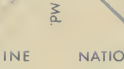
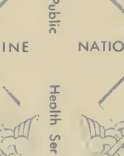
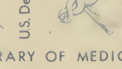
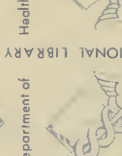
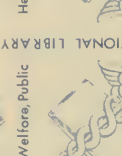
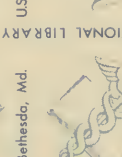
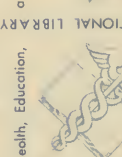
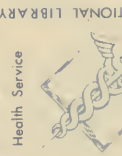
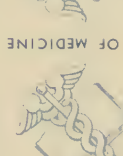
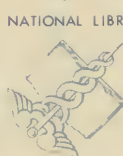
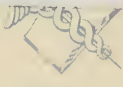
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Clinical Roentgenology

OF

Diseases of the Chest

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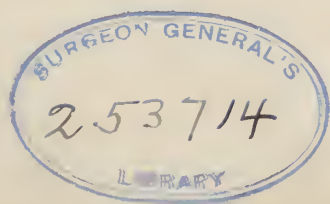
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DEDICATED
TO THE MEMORY OF
EUGENE WILSON CALDWELL, B. S. M. D.
PIONEER AMERICAN ROENTGENOLOGIST
AND MARTYR TO THE SCIENCE

Preface

In the preparation of this work, we have had two objects in mind, first, an exposition of the Roentgen features of thoracic diseases and secondly, their correlation with such clinical facts as are necessary to an intelligent Roentgen interpretation. It is therefore hoped that the following pages will commend themselves no less to the clinician than to the Roentgenologist. We have been heartened to our task, in spite of the difficulties attending it, by the lack of any adequate treatise on this subject in the English language.

In no branch of medicine has the clinical application of the Roentgen ray yielded more gratifying results than in diseases of the chest. It has effected a revolution in thoracic diagnosis and, when properly employed, has eliminated much of the uncertainty inherent in the older methods of examination.

It is not our purpose, however, in these prefatory remarks, unduly to glorify the Roentgen examination; we may safely leave its achievements, illustrated in the following pages, to speak for themselves. Rather is it our wish, among other things, to emphasize the importance of a correlation of the Roentgen with the clinical examination, which is nowhere more vital to correct diagnosis than in diseases of the chest.

The question arises—To whom shall we look for such a correlation, to the clinician or to the Roentgenologist? Unfortunately, the latter is rarely a capable clinician and his medical inexperience is fraught with grave consequences in the diagnosis of thoracic disease. On the other hand, the clinician is, as a rule, poorly trained in the technique of Roentgenology and in the interpretation of its findings. Technical excellence is perhaps more essential in the examination of the chest than of any other part of the body. A slight fuzziness in outline or a distortion of the shadows is fatal in roentgenograms of the lungs, in which dependence is placed on the faintest of shadows and the sharpest of lines for the differentiation between normal and pathological conditions.

For these reasons, the ideal of a clinician-roentgenologist can rarely be attained and the difficulties arising from the diversity of their training, can, for the present, only be overcome by a close co-operation of the clinician and roentgenologist in arriving at a diagnosis. Such co-operation is especially important in surgical diseases of the chest where an interpretation of the plates in the light of the clinical facts must often determine the judgment of the surgeon as to his procedures.

It may not be amiss to insert here a few remarks on Roentgen technique which have purposely been omitted from the text. In order to obtain on the plate a faithful reproduction of the fine linear markings of the lung, it is necessary that the patient be placed at a considerable distance from the tube. At lesser distances, especially at the conventional 28 inches, these markings are apt to suffer enlargement and distortion which render accurate diagnosis difficult or impossible. For practical

purposes, having due regard for the time of exposure, we have found that a distance of four feet will meet the requirements of the average case. Of no less importance is the position of the patient, which should be upright whenever possible. The prone position, with the resultant upward displacement of the diaphragm, engorgement of the lungs and interference with breathing, causes such an exaggeration of the lung markings, that the plates are of little value for diagnosis.

The use of intensifying screens, except with very sick patients who cannot control their breathing or for tele-roentgenograms, has seemed to us a distinct disadvantage in chest work. The lack of perfect contact and the defects which develop in the screens, result in a hazy picture which cannot compare with the clearness and definition of a good plate.

To obtain the necessary penetration of the ray at the distance which we have adopted, it has been found advantageous to employ a spark gap of 7 to 8½ inches at 40-50 milliamperes. This necessitates the use of gas tubes, as the type of Coolidge tube which can operate at this setting emits so many parasitic radiations from the anticathode stem, that the image loses its definition. The high spark gap is especially adapted to the Roentgen examination of the lungs, as the penetration of the ray is facilitated by their air content and contrast is afforded by the soft tissues of the thorax.

In regard to the purely clinical side of this work, no attempt has been made to present the clinical aspect of diseases of the chest in a complete or systematic manner. That would be foreign to its purpose. We have rather sought to emphasize those features of the clinical history, physical signs and pathology which are necessary to an intelligent interpretation of the plates. On the other hand, we have tried to present the facts of Roentgenology as completely as possible and in accordance with the most enlightened opinion in respect of interpretation. Some phases of the Roentgen diagnosis of chest disease, notably that of pulmonary tuberculosis, are still the subject of controversy. In regard to it, we have not hesitated to express our personal views, believing as we do, that they are in accord with the best thought and best harmonize with what we know of its pathology.

Nor have we hesitated to point out, in numerous instances, the limitations of the Roentgen ray in respect of diagnosis. It must always be borne in mind that a Roentgen plate is, after all, only a conglomerate of shadows, the arrangement of which, though usually characteristic of definite diseases, is occasionally susceptible of different interpretations.

It is a pleasure to acknowledge the help of those who have co-operated with us in the preparation of this work. We have been fortunate in securing the aid and advice of Mr. Walker of the Walker Engraving Company in the making of the half tones and of Mr. S. W. Nourse whose skillful photographic work helped materially in the reproduction of the original negatives. Finally, we are indebted to the publishers, The Southworth Co. without whose generous helpfulness this work would not have been possible.

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- 21a. Detail of fig. 21.
22. Early tuberculous infiltration, infraclavicular.
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24. Early tuberculosis with pneumonic exudate.
- 24a. Detail of fig. 24.
25. Early tuberculosis, showing tubercles.
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27. Early tuberculosis with a variety of infiltrations.
28. Earliest tuberculous infiltration.
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33. Healing infiltrations of early tuberculosis.
34. Fibrous network in healing tuberculosis.
- 34a. Detail of fig. 34.
35. Fibrosis of healed early lesion in infraclavicular region.

36. Scar of healed tuberculosis.
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185. Chronic lung abscess simulating lung tumor.
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190. Actinomyces of lung and pleura.
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- 197, 198. Gumma at root of left lung; pressure on bronchus, with secondary suppuration. Before and after treatment.
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203. Carcinoma of left upper lobe; phrenic paralysis.
- 204, 205. Adeno-carcinoma of right bronchus with subsequent invasion of both lungs.
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207. Adeno-carcinoma of right upper lobe with necrosis; metastases in left lung.
208. Infiltrating carcinoma of lung with bronchial occlusion.
209. Alveolar carcinoma of lung, with secondary abscess formation.
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212. Carcinoma of lung with clinical picture of lung abscess.
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220. Multiple metastases in lung, secondary to fascial sarcoma of thigh.
221. Metastatic sarcoma.
222. Metastases in lung with sarcoma of testis.
223. Metastatic carcinoma of lung, primary in stomach.
224. Large metastatic carcinoma of lung.
225. Metastatic carcinoma secondary to thyroid tumor.
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230. Miliary carcinosis of lung; perforation of cortical tumors with localized sero-pneumothorax.
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- 246. Encapsulated apical empyema with involvement of dependent part of chest.
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- 271. Diagram of figure 270.
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- 274. Large effusion between the right upper and lower lobes.
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279. Multilocular encapsulated pyo-pneumothorax; patient in lateral recumbent position showing three fluid levels.
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285. Complete pneumothorax, depression of diaphragm and compression of lung.
286. Spontaneous pneumothorax with miliary tuberculosis of the lungs.
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288. Pneumothorax under tension, with extensive bilateral tuberculosis.
289. Pyopneumothorax.
290. Pneumopericardium complicating pyopneumothorax.
291. Pneumothorax encapsulated by adhesions, simulating cavities.
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307. Chronic mediastinitis secondary to multiple bronchiectases.
308. Chronic mediastinitis secondary to fibroid tuberculosis; oesophageal obstruction.

309. Chronic mediastinitis with diaphragmatic adhesion, secondary to aneurysm of aorta.
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312. Large mediastinal tumor encasing the heart.
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314. Angio-sarcoma of mediastinum.
315. Infiltrating mediastinal lympho-sarcoma associated with Hodgkin's disease.
316. Infiltrating mediastinal tumor.
317. Mediastinal tumor infiltrating right upper lobe.
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319. Metastatic carcinoma of mediastinal lymph nodes, secondary to bronchial tumor; clinical picture of heart failure.
320. Large unilateral mediastinal tumor; cardiac displacement.
321. Large lobulated mediastinal tumor.
322. Leukemic mediastinal tumor; pleural effusion.
323. Mediastinal tumor with pulmonary metastases.
324. Mediastinal sarcoma, resembling aneurysm of the aorta.
325. Two stages in the growth of a tumor of the superior mediastinum.
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329. Small substernal thyroid gland.
330. Cervical goiter with intrathoracic extension.
331. Intrathoracic goiter with calcification.
332. Large substernal goiter with tracheal displacement.
333. Adeno-carcinoma of the isthmus of the thyroid.
334. Unilateral intrathoracic goiter; compression of trachea.
335. Calcification of right lobe of thyroid.
- 336, 337. Calcification of old colloid goiter of the isthmus; dorso-ventral and oblique view.
338. Variation of the normal mediastinum in infant in deep inspiration and expiration.
339. Enlarged thymus in infant 12 hours old.
340. Large thymus showing typical keystone shape.
341. Large thymus in 19 day old infant, without symptoms.
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342. Tumor of thymus; thymoma; pleural effusion.
343. Dermoid cyst of the mediastinum.
344. Dermoid cyst of the mediastinum.
345. Fusiform aneurysm of the ascending aorta.

- 346. Fusiform aneurysm with calcification of the transverse arch.
- 347. Large fusiform aneurysm of the entire aortic arch, involving also the innominate artery; calcification and periarteritis.
- 348, 349. Sacculated aneurysm of the ascending arch, showing growth after three years.
- 350. Sacculated aneurysm of the beginning of the ascending arch.
- 351. Sacculated aneurysm of the ascending and transverse arch.
- 352. Sacculated aneurysm of ascending and descending arch of aorta; tracheal displacement.
- 353. Sacculated aneurysm of the ascending and descending arch.
- 354. Large aneurysm of the transverse arch.
- 355. Aneurysm of the transverse arch.
- 356. Large aneurysm of the transverse arch.
- 357. Fusiform aneurysm of the descending arch of the aorta.
- 358. Perforating aneurysm of the ascending arch.
- 359. Globular dilatation of the entire arch of the aorta.
- 360. Oblique view of aneurysm of the aorta, show compression of the trachea and oesophagus.
- 361. Dynamic dilatation of the aorta in case of luetic aortitis with aortic insufficiency.
- 362. Aneurysm of the innominate artery.
- 363. Acute bronchial lymph-adenitis secondary to retropharyngeal abscess.
- 364. Hyperplasia and induration of bronchial lymph nodes.
- 365. Calcification of tuberculous bronchial lymph nodes.
- 366. Enlargement of right paratracheal lymph nodes in syphilis.
- 367. Enlargement of bronchial nodes in a syphilitic.
- 368. Enlargement of upper mediastinal lymph nodes in acute myeloblastic leukemia.
- 369. Enlargement of bronchial lymph nodes in acute leukemia.
- 370. Enlargement of bronchial lymph nodes and infiltration of lung in chronic lymphatic leukemia.
- 371. Mediastinal tumor in Hodgkin's disease.
- 372. Mediastinal tumor in Hodgkin's disease.
- 373. Unusual infiltrating form of Hodgkin's disease.
- 374. Hodgkin's disease; diffuse infiltration of both lungs.
- 375. Enlargement of bronchial lymph nodes in Hodgkin's disease; infiltrations in the lungs.
- 376. Hodgkin's disease; characteristic enlargement of the bronchial and paratracheal lymph nodes.
- 377. Hodgkin's disease; very large paratracheal nodes, diffuse infiltration of right lower lobe.
- 378. Hodgkin's disease; characteristic enlargement of the bronchial and paratracheal lymph nodes.

- 380 Prominence of mesial portion of right diaphragm, due to elevation of the central tendon.
- 381. Subphrenic abscess; dry pleurisy at right base.
- 382. Subphrenic abscess on left side.
- 383. Subphrenic infected hematoma.
- 384, 385. Subphrenic gas-abscess secondary to duodenal perforation.
- 386. Large subphrenic gas abscess.
- 387. Elevation of left half of diaphragm by echinococcus cyst of the spleen.
- 388. Eventration of the diaphragm, left side, with displacement of the heart.
- 389. Eventration of the diaphragm, left side.
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- 391. Hernia of the stomach through bullet wound of diaphragm.
- 392. Eventration of the diaphragm distinguished from diaphragmatic hernia by means of pneumoperitoneum.
- 393. Chronic atonic dilatation of the stomach with elevation of the diaphragm.
- 394. Induced pneumoperitoneum showing outlines of diaphragm, liver and spleen.
- 395. Subphrenic pneumoperitoneum following perforated gastric ulcer.
- 396. Accidental pneumoperitoneum following artificial pneumothorax.
- 397. Ptosis of the liver, with interposition of the colon between it and the diaphragm.
- 398. Pointed diaphragmatic adhesion with chronic pulmonary tuberculosis.
- 399. Fixation and distortion of the diaphragm by adhesions following empyema.
- 400. Fixation of the left leaf of the diaphragm by adhesions.
- 401, 402. Pneumonia of the right lower lobe, followed by adhesions and deformity of the diaphragm.
- 403. Postoperative mesial pyo-pneumothorax.
- 404. Thickening of parietal and visceral pleura following empyema; gradual expansion of lung.
- 405. Deformity following extrapleural compression of chest for chronic empyema; thickening of pleura.
- 406. Persisting empyema sinus, with retention of pus.
- 407. Chronic bilocular empyema sinus injected with bismuth.
- 408. Chronic empyema sinus injected with bismuth.
- 409. Thoracic deformity resulting from decortication of lung for chronic empyema.
- 410. Extrapleural compression of residual lung abscess by means of injected paraffin oil.

- 411. Extrapleural compression of residual lung abscess by means rubber balloon.
- 412. Maldevelopment of right first rib.
- 413. Fusion of left first and second ribs.
- 414. Deformity of right half of chest due to aplasia of upper ribs.
- 415. Bifid sternal end of left third rib.
- 416. Sarcoma of right seventh rib; dorso-ventral and oblique views to distinguish from pleural disease.
- 417. Multiple rib sarcomata.
- 418, 419. Sarcoma of right seventh rib, extensively invading soft tissues, before and after operation.
- 420. Osteoma of sternal end of second left rib.
- 421. Multiple myeloma of ribs, spine and shoulder girdle.
- 422. Sarcoma of cervical spine and adjacent ribs, simulating a mediastinal tumor.
- 423. Pott's abscess in lower dorsal region.
- 424. Pott's abscess almost obscured by the heart.
- 425. Deformity of the chest with kyphosis.
- 426. Deformity of the chest with kyphosis.
- 427. Deformity of the chest with kyphoscoliosis.
- 428. Carcinoma of the breast casting shadow simulating intrathoracic disease.
- 429. Calcified cervical and supraclavicular lymph nodes overlying the apical lung fields.
- 430. Axillary lymph nodes in Hodgkin's disease overlying the pulmonary field in the right axilla.

SECTION I

The Normal Lung

CHAPTER I

The Normal Lung

A satisfactory description of the normal lung is very difficult owing to the great variation in the shape and arrangement of the pulmonary shadows. There is, strictly speaking, no normal lung type, because no two lungs are alike anatomically. This difference is reflected in their Roentgen appearance and it is only through a study of a large number of plates that the Roentgenologist learns to set the dividing line between the normal and the pathological lung.

If we consider for a moment the anatomic variations of the normal lung, their influence on the appearance of the Roentgen plate will be apparent. In no two individuals are the size and the arrangement of the pulmonary vessels the same and therefore the shadows which they cast will constitute a network of various density and pattern. The amount of connective tissue in the lung will also modify the appearance of the plate. With increasing age peri-vascular and peri-bronchial deposits of connective tissue together with anthracosis will manifest themselves by an increased density of the shadows.

Aside from inherent anatomic variations, we must keep in mind the numerous deviations from the healthy normal lung which arise from the inflammations to which we are subject, the effects of which are manifest on the plate. It happens therefore, that in our search for a normal lung plate we will in the vast majority of cases be confronted by the evidences of previous disease, which will commonly be found at the roots of the lungs and occasionally within the parenchyma. It is evident that the Roentgenologist in his study of the plates for signs of pulmonary disease must not place an undue emphasis on these changes which are of almost universal occurrence; he must learn that they are not associated with clinical disease and he must be prepared to regard such lungs as normal, at least in the Roentgen understanding of the term.

The character of the Roentgen plate is notably influenced by the technique employed in its production. When the distance of the tube from the patient is short, the pulmonary shadows are exaggerated in size and acquire a haziness which interferes with their interpretation. For careful work it is also necessary to make the examination in the upright position. Only thus it is possible to avoid engorgement of the pulmonary vessels and a corresponding blurring of their shadows, which vitiate the Roentgen examination when the patient is in the

horizontal position. If any conclusions of value are to be derived from a Roentgen examination of the lungs it is absolutely necessary that such extrinsic sources of error be avoided. For this reason, the examination should whenever possible, be made with the patient in the upright position and at as great a distance from the tube as is consistent with a thorough exposure.

A description of the features of the normal lung may conveniently be divided into two parts. The first deals with the aerated lung fields in which are included the parenchyma, the bronchi, the blood vessels and the lymphatics. The second is concerned with the lung roots.



FIGURE 1

Normal lung of a young adult woman. Root shadows are approximately normal.
Exposure at 5 feet, upright position.

THE AERATED LUNG FIELDS

The lung fields comprise a space enclosed by the bony thorax and the diaphragm; it is divided into two parts by the heart and the mediastinum. Each of these fields is traversed by a network of shadows which are produced by the bronchi, blood vessels and lymphatics and by the small amount of connective tissue about them. They are capable of casting recognizable shadows by virtue of the air containing lungs in which they lie, which afford them the necessary contrast. (Fig. 1 and Fig. 2.)



FIGURE 2
Normal lung of a young adult man.

These shadows are worthy of closer description. It will be noted that they spread out radially from the root of the lung to the periphery and that they correspond in their arrangement to the branching of the bronchial or the vascular tree. Because these so-called markings of the lungs are a composite picture in the one plane of the plate, of numberless structures which lie in different planes in the lung, this net work is not a continuous and distinct one but is fragmentary and indistinct in outline. Only at the roots of the lungs where the vessels and bronchi are larger may we speak of the shadows as corresponding to a single and recognizable structure.

The anatomical basis of the lung markings has long been a cause of dispute. They have variously been ascribed to the bronchi, to the blood vessels and to both of them together. At the present time, opinion, based both on experimental and clinical grounds inclines to the belief that the lung markings owe their origin in varying degrees to both the blood vessels and the bronchi. At the roots, the trachea and large bronchi contribute perhaps to a considerable degree to the production of the shadows, although even here the blood vessels play the greater role. As one approaches the periphery, the blood-filled vessels have a greater and greater share in the production of the shadows until at the surface of the lung the fine mottling is probably exclusively due to the small branching vessels. The markings become progressively smaller as they approach the periphery so that the lung acquires a mottled or marbled appearance and in the average individual they are no longer visible in the apices and in the axillary region of the chest. In these locations the lung has a homogeneous gray or black appearance depending upon the intensity of the exposure.

There are certain portions of the lung in which the markings are normally more prominent than in others; for example, in the para-vertebral portion of the upper lobes there are numerous coarse strands which extend vertically upward from the hilum to the clavicle and at the bases also there are large branching shadows of the pulmonary vessels which extend from the roots of the lungs along the right and left borders of the heart to the diaphragm. The significance of the latter shadows will be discussed at greater length in another connection.

THE LUNG ROOTS

The root or hilum of the lung is represented on the plate by an irregular shadow situated near the mid-line along the borders of the heart. Within it are merged the shadows of the large pulmonary vessels, the bronchi, lymph nodes and the connective tissue which surrounds these structures. With such heterogeneous components, it is obvious that the hilum shadows can have no definite or constant shape and that there is room for considerable variation in their Roentgen appearance.

The interpretation of the hilum shadows has given rise to much confusion from a failure properly to appraise the pathological changes which are so frequently present at the roots of the lungs. Pathological studies show the almost universal occurrence either of hyperplasia and anthracosis of the bronchial lymph nodes and also caseation and calcification resulting from tuberculous involvement of these nodes. In addition there is often an increase in the connective tissue about them. The root shadows which we see in adults therefore practically never reflect a normal condition. Nevertheless, so common are these changes at the root and so rarely are they responsible for any symptoms that no clinical importance need be attached to them. It is particularly unfortunate that an enlargement of the shadows at the roots of the lungs has so often been made a basis for the diagnosis of pulmonary tuberculosis by Roentgenologists.

In the normal hilum the shadows are of moderate density, shade off almost imperceptibly into the lung tissue and the markings of the large vessels and bronchi are faintly indicated within it. The normal lymph nodes are not distinguishable as they are merged in the general shadow mass. Such a hilum for the reasons given above, is rarely encountered. In the average adult the hilum extends further into the lung fields, sometimes for a considerable distance. The shadows are denser and some of them, representing hyperplastic lymph nodes, are discrete and distinct in outline. Anthracotic and calcified nodes and those which are caseous cast very dense clearly outlined shadows. Irregular dense shadows, sometimes in ring form may be due to deposits of lime in tuberculous nodes. Although the bronchi are as a rule not visible, they may occasionally be seen in outline at the root of the lung if the tissue in this region is infiltrated. Thus they may be seen as small annular shadows, or in double contour, depending on the incidence of the Roentgen Ray.

An increase in the size of the hilum may be apparent rather than real. Thus, a narrow heart or one which is median in position permits more of the hilum to be seen and it may therefore appear to be larger. In cases of emphysema or chronic bronchitis, aside from the actual enlargement of the lymph nodes, the roots may appear to be larger and denser because of the vivid contrast furnished by the over-aerated lungs.

The individual groups of lymph nodes are only distinguishable in a general way. The tracheo-bronchial nodes are covered by the heart and are only demonstrable to a limited degree by an oblique examination. Only the broncho-pulmonary and the bronchial nodes are visible where they are not obscured by the heart shadow. Occasionally the para-tracheal nodes especially on the right side are seen when they are enlarged by disease.

Although the hilum of the lung has up to recent times loomed large in the reports of Roentgenologists, experience has taught that inferences drawn from it are of little value in clinical diagnosis. In the vast majority of cases, particularly in adults, the changes which are discovered at the roots have no clinical significance and aside from noting their presence, they are best disregarded.

AERATION OF THE LUNGS

The aeration of the lungs is a variable element which is arbitrarily estimated by the photographic character of the plate. For this reason it is scarcely necessary to state that in determining the air content of the lungs one must be informed as to the intensity of the exposure. It is for example, hazardous to regard a lung as emphysematous because the plate looks abnormally black. Note must be taken of the bones and the soft tissues in order to determine whether the chest has been overexposed. Aside from this, the apparent aeration of the lungs is affected by the thickness of the chest wall and also by the depth of lung tissue which the rays traverse. The upper and axillary portions of the lungs which are covered by muscle and fat will not appear as well aerated as the remaining portions and due note must be taken of the shadows cast by the pectoral muscles in men and by the breasts in women. At both bases, along the border of the heart, the presence of large blood vessels decreases the apparent aeration of the lungs.

Particular caution must be exercised in estimating the aeration of the apices. Well developed supraclavicular muscles and deposits of fat above the clavicle added to the relatively small mass of air containing tissue at the apices will make them appear poorly aerated. The shadows cast by cervical ribs and fleshy neck muscles will have the same effect. It must also be remembered that in some cases, owing to the conformation of the chest it is not possible to bring the apex in close apposition to the plate and for this reason it will appear to be less aerated. On the other hand, one of the best aerated portions of the lung is situated just above the diaphragm in its axillary portion. There is here great depth of lung tissue which is not covered by excessive muscle or fat. It will be evident that only when these normal variations both in the real and the apparent aeration are kept in mind, may reliable conclusions be drawn from a difference in the illumination of any portion of the plate.

On fluoroscopic examination of the lungs one may observe the influence of respiration on their air content. Very little change is noted on inspiration in any portion of the lungs, except in that just above the diaphragm. At this point the lungs have their greatest expansion and light up considerably. In the apices, on the contrary, there is little or

no respiratory change in the aeration. Only on coughing there is noted a sudden distention of the apex associated with a distinct increase in aeration.

In addition to the shadows which are cast by the lungs themselves, it must be borne in mind that there are numerous other shadows arising from the tissues of the chest wall and the other thoracic viscera which are projected over those of the lungs. With these shadows, which may at times cause difficulties in interpretation, the Roentgenologist must become familiar if he is to avoid error. It is particularly in the upper aperture of the chest that confusion is apt to arise because in this situation the aorta, the innominate vessels, the upper ribs and the trachea may produce a complicated shadow mass. Occasionally also, unique shadows, as in figure 3 are seen at the apex, whose significance is not clear.



FIGURE 3

Unique falciform shadow at right apex. This shadow is found in a small percentage of patients and is always seen on the right side.

It appears to have no clinical significance.

SECTION II

The Pulmonary Vessels and Circulation

CHAPTER II

The Pulmonary Vessels and Circulation

In our discussion of the anatomical basis of the lung markings, we have indicated to what extent the pulmonary vessels are responsible for their production. Although all the component parts of the lung combine to produce the lung arborizations, the blood vessels undoubtedly cast the major portion of the shadows. We are therefore justified, to some extent, in estimating the vascularity of the lungs by the intensity and width of its linear markings.

Unfortunately we have no definite standard by which to judge deviations from the normal vascularity. There are numberless variations in the size of the lung markings which have no relation to pathologic alterations in the filling of its blood vessels. Some of these variations are intrinsic in the lung and are due to changes resulting from previous infection, whereas others depend on such extraneous factors as the technique of the exposure and the position of the patient. We must accordingly keep these sources of error in mind when we seek to interpret the Roentgen shadows in terms of an altered vascularity of the lungs.

In the Roentgen study of the circulatory changes in the lungs, it will be convenient to discuss first those which affect the intra-pulmonary blood vessels and secondly those which involve only the conus of the pulmonary artery.

PASSIVE CONGESTION

The commonest cause of an increased vascular filling of the lung is the congestion of heart disease, which occurs in its most pronounced form in mitral lesions. In the early, compensated stage of this disease, no change is to be noted in the size of the vessel markings. With a failure of the heart muscle and dilatation of its chambers, stasis in the pulmonary veins ensues, which manifests itself on the Roentgen plate in various ways. In the first place, the large vascular trunks which issue from the heart and normally comprise the hilum, become enlarged and are seen as wide branching shadows which extend toward the periphery of the lung. (Fig. 4.) Two groups of vessels are especially prominent; one extends upward in the para-vertebral region to the apices and a second, larger one, traverses the lower lobes along the borders of the heart. A second change in the pulmonary fields results from an engorgement of the smaller vessels within the parenchyma of the lung. The



FIGURE 4

Enlargement of the large branches of the pulmonary vessels in mitral disease.

lungs appear poorly aerated and the dilatation of the vessels produces a diffuse mottling in which the normal appearance of the bifurcating vessels is lost. (Fig. 5.) This mottling is usually more noticeable in the lower lobes. In the later stages of decompensation, especially in the presence of some pulmonary edema, the pulmonary fields become quite hazy and on fluoroscopic examination the normal increase in aeration during deep inspiration may be entirely lacking.

The Roentgen evidences of pulmonary congestion, as we have described them, are in themselves of minor importance. They arrest our attention however because the question will occasionally arise, whether, in addition to the valvular lesion, the patient also has pulmonary tuberculosis. Rales and dulness are common accompaniments of pulmonary congestion and infarction of the lung may be associated with



FIGURE 5

General mottling of the lungs due to the passive congestion of mitral disease.

frequent hemoptyses and fever. The clinical picture may therefore closely resemble tuberculosis. It is therefore important, when we read the plate in cases of heart disease to recognize the changes which are due to vascular engorgement and not to confuse them with the infiltrations of tuberculosis. The similarity between congested lungs and tuberculosis may in some cases be so close that the distinction between them cannot be made with certainty. This is particularly true of mitral

disease in which the blood vessels in the para-vertebral part of the upper lobes are often enlarged and nodular so that they closely resemble tuberculous infiltrations.

In coming to a decision in these cases we are fortunately aided by the knowledge that tuberculosis of the lungs rarely complicates a valvular defect. The coincidence of these two diseases is so uncommon that in a case of chronic endocarditis the Roentgenologist will wisely interpret the abnormal pulmonary shadows as the result of pulmonary congestion, unless there are indubitable infiltrations in the periphery of the lung. Fortunately a mitral heart presents a characteristic configuration on the Roentgen plate which cannot easily be mistaken and its discovery will lend assurance to our interpretation of the questionable shadows.

A passive congestion of extreme degree may occasionally be seen in the lungs in cases of pressure on the roots by mediastinal new growths. (Fig. 319.)

ABNORMALITIES OF THE PULMONARY CONUS

An abnormal prominence of the conus of the pulmonary artery is regularly found in cases of mitral disease and it contributes, together with the enlarged left auricle, to the heart silhouette which is so characteristic of this valvular lesion. It is questionable whether the artery is in most cases as much dilated as it is displaced upward by the dilated and hypertrophied right ventricle. Unlike the aorta, little or no pulsation is visible in the artery, even when it is much enlarged. It is not improbable that the rough systolic murmurs which are so often heard in the region of the pulmonary artery in cases of advanced mitral disease, have their origin in this vessel, whose enlargement and consequent nearness to the chest wall will favor the transmission of adventitious sounds generated within it.

Next to mitral lesions, the commonest cause of a dilatation of the pulmonary artery, is congenital heart disease. It may result from an anomalous development of the vessel itself or it may be associated with congenital defects in other portions of the heart.

PERSISTENT DUCTUS BOTALLI is by far the most common congenital lesion in which the pulmonary artery is much dilated. This vessel, being the recipient of blood from both the right ventricle and from the aorta through the ductus arteriosus may assume unusually large proportions and exhibits a forcible systolic pulsation of such amplitude as is rarely to be seen in any other condition. In young children the enlargement of the vessel is usually slight; with the passage of years however, it gradually becomes greater until in early adult life,

coincident with the development of the continuous, so-called "machinery murmur," the characteristic prominence of the pulmonary artery will be evolved (Fig. 6). At this stage of the disease, there will usually be



FIGURE 6

Enlargement of the conus of the pulmonary artery with open Ductus Botalli.

found on physical examination the area of para-sternal dulness on the left side, which Gerhardt early associated with a dilatation of this vessel.

It must be borne in mind however, in interpreting an enlargement of the pulmonary artery that a mal-development and hypoplasia of this vessel may accompany the most diverse congenital malformations, aside from a persistent Ductus Botalli. The thin, distensible artery yields to the pressure of the blood within it so that it may cast an unusually wide shadow on the plate. For this reason, it is safe to attribute the enlargement of the pulmonary artery to a persistent ductus, only when it can be brought into harmony with the more or less characteristic physical signs which are found in this condition.

The pulmonary artery may be congenitally larger from an unequal subdivision of the primitive conus arteriosus into a larger pulmonary and a smaller aortic portion. This anomaly need in no way affect the efficiency of the heart or the circulation and it may remain clinically latent. It was thus accidentally discovered in a middle aged man who ultimately died of suppurative pyelophlebitis. In figure 7 there is to be



FIGURE 7

Congenital enlargement of the pulmonary artery and its branches. Hypoplasia of aorta. The shadows at the roots of the lungs are the large branches of the pulmonary artery; one on the right side is seen in cross section.

seen an enormous enlargement of the pulmonary artery which pulsated forcibly. At the root of the right lung, its main branches, which were also congenitally larger, cast large, dense shadows which extend far into the pulmonary fields. The aorta is hypoplastic and its shadow barely extends beyond the spine. There were no murmurs, dyspnoea, or cyanosis.

Syphilis must be numbered among the rarer causes of an enlarged pulmonary artery. An aneurysm of this vessel does not signalize its presence by the striking symptoms of aortic disease nor are these symptoms of such a character as to cause an investigation of the pulmonary artery. It therefore happens that this rare affection may be discovered

on the plate incidentally during a routine examination of the heart or lungs. Just as to the clinician the region of the pulmonary valves is the area of cardiac romance, so to the Roentgenologist this portion of the cardiac contour is replete with surprising disclosures of which the clinical examination supplies no inkling. The physician who has exhausted the resources of physical examination without finding any definite evidence of disease of the pulmonary artery, may be pardoned a feeling of astonishment when the Roentgen plate reveals an enormous aneurysm as shown in figure 8.



FIGURE 8

Aneurysm of the pulmonary artery.

Minor grades of dilatation are observed when the pulmonary artery is the seat of athero-sclerosis. A similar pathological change is occasionally found in emphysema and some observers see a significant relation in certain cases between the atheroma and dilatation of the pulmonary artery and the accompanying dyspnoea and cyanosis.

It remains for us to consider certain types of real and others of apparent enlargement of the pulmonary artery, which have apparently no clinical significance. For example, in figure 9 is illustrated an abnormal prominence of the vessel, unassociated with any cardiac or

THE PULMONARY VESSELS AND CIRCULATION



FIGURE 9

Enlargement of the pulmonary conus.

pulmonary disease, the significance of which we are at a loss to explain. At times these patients exhibit an accentuation of the second pulmonic sound and if the heart is overacting, a pulsation may be visible in the second left intercostal space. Clinicians have in certain cases sought to explain these phenomena as a result of the retraction of the edge of the lung due to tuberculosis, which permits a more ready transmission of the pulmonary second sound to the chest wall. Although this may be the mechanism in some cases, it is not true of all and we must concede the existence of a dilated pulmonary artery in some of these patients without coincident disease of the lung. The enlarged pulmonary artery here represents only an interesting variant from the normal.

Finally we must take note on the Roentgen plate of an enlargement of the pulmonary artery, commonly observed in hypoplastic and true drop hearts, which is however apparent rather than real. The drop heart represents probably an earlier type in the phylogenetic scale in

which this organ is situated centrally nearer the anterior chest wall and is also rotated on its vertical axis. This movement brings the pulmonary artery into greater prominence on the Roentgen plate without any actual enlargement. It is also calculated to increase the intensity of the second pulmonic sound. As pulmonary tuberculosis frequently develops in the asthenic individual with a drop heart, we have here a possible explanation for the accentuated basal sounds which are often noted in pulmonary disease.

Occasions will arise when it will be difficult to distinguish the shadow of a pulmonary artery from that of a neoplasm of the lung or mediastinum. How closely the shadows of these two diverse conditions can simulate each other will be evident from a study of the mediastinal tumor illustrated in figure 326. It will be necessary in such a case to have recourse to the fluoroscopic examination in order to determine the presence of a pulsation and particularly to view the abnormal mass in the oblique position. It is characteristic of an enlarged pulmonary artery to become more prominent in the first oblique position, whereas most neoplasms in this situation will wholly or partly disappear from view.



FIGURE 10
Infarct of the left lung.

PULMONARY INFARCT

Infarcts of the lung cast recognizable shadows on the plate only when they are large. It is a common experience to find at autopsy numerous small infarcts of which the Roentgen examination gave us no evidence during life. In their typical forms they appear as large triangular shadows, with their bases toward the axilla, which may occupy a large part of a lobe. (Fig. 10.) On the other hand, the lung about them may be in such a state of inflammatory reaction that the resulting shadow may be indistinguishable from that of a pneumonic



FIGURE 11

Arterio-venous aneurysm of the pulmonary artery following lung abscess.

process. It is important, however, not to regard every extensive shadow in the lung, occurring in cardiac disease, as of embolic origin. The clinician is well aware of the frequent incidence of true pneumonias, due to the virus of rheumatism or allied micro-organisms, during the course of chronic endocarditis. They are often atypical in their clinical course and puzzling in their physical manifestations; they may, like infarcts, be a terminal event in the case and unfortunately the Roentgen shadows will leave us in the dark as to their true nature.

In conclusion it may not be amiss to illustrate the following rare circulatory condition which exemplifies the versatility of the Roentgen examination in thoracic diagnosis. Figure 11 represents an arterio-venous aneurysm which was clinically symptomless, following a lung abscess. The circular shadow in the left lung was found at autopsy to be an encapsulated blood cyst which communicated on one side with the pulmonary artery and on the other with the pulmonary vein. The abscess had completely healed.

SECTION III

The Trachea and Bronchi

CHAPTER III

The Trachea

In a thoroughly exposed plate, the lumen of the trachea may be seen as an air space against the spine which forms its background. It traverses the upper part of the chest, usually in the midline, although occasionally it deviates slightly to the right or to the left. At the level of the fourth dorsal vertebra it bifurcates into its two branches. Its calibre is generally uniform and varies in width with the size of the individual. There are several points at which it may be slightly narrowed notably below the cricoid cartilage and also at the bifurcation. The topographical relations of the trachea and bronchi in the living subject are graphically set forth in figure 15 in which they are outlined by aspirated barium.

The wall of the trachea casts no shadow on the plate except in its lower portion where, on the right side, it may encroach on the pulmonary field, especially when the head is turned. The tracheal rings are invisible, unless, as in older subjects they are calcified. They are best seen in the lateral position. (Figure 12.)

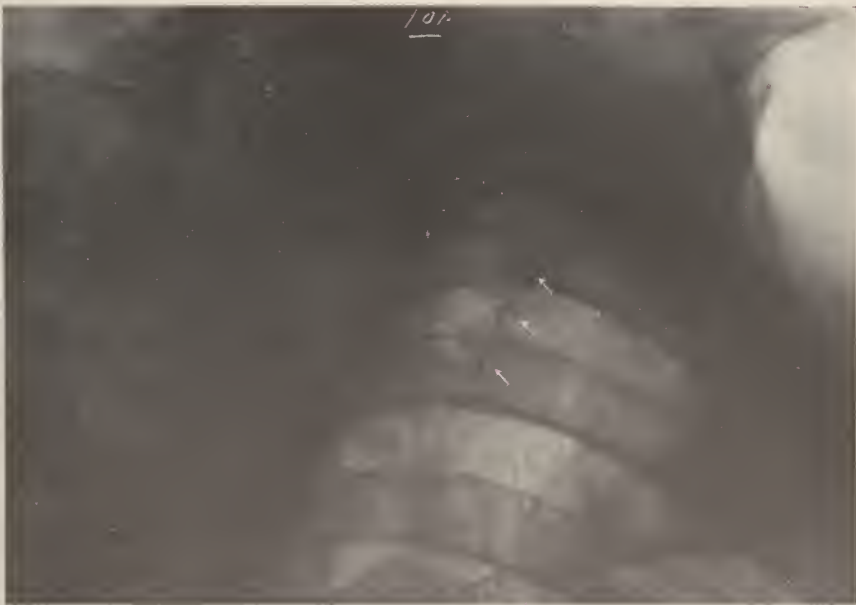


FIGURE 12
Calcified tracheal cartilages, seen in the oblique position.

Study of the trachea by means of the Roentgen ray does not derive its interest from any intrinsic diseases of this structure, which are very uncommon. Its importance depends rather on its position in the thorax where it comes into relation with a number of organs, disease of which is often reflected in an altered configuration of the trachea. The latter, together with the superior mediastinum constitutes a movable and non-rigid structure which readily yields to any abnormal pressure which may be applied to it. The numerous causes for such pressure which may arise in the mediastinum, lungs and pleura may result in a displacement of the trachea or its compression or both.

DISPLACEMENT OF THE TRACHEA

The commonest cause of a displacement of the trachea is pulmonary tuberculosis, particularly of the upper lobes. The causes of this displacement are probably various. In the first place a diminution in the volume and respiratory movement of the affected lung, due either to infiltration or atelectasis may, even in early tuberculosis upset the balance of pressure on the two sides of the trachea; consequently the distention of the sound lung pushes it to the affected side. This will account for the slight deviation of the trachea in cases of early tuberculosis, in which there is no evidence of fibrosis or traction.

The more marked degrees of tracheal malposition are associated with the fibrosis of older lesions. In these cases, the trachea, being relatively fixed at two points, namely, at the cricoid cartilage and at the bifurcation, is drawn to the affected side and also takes a tortuous course through the upper chest. This is almost a regular appearance in advanced pulmonary tuberculosis. In cases of bilateral disease, it indicates the side of the older lesion, as the trachea is drawn over to the side of the original process. Here will be found the evidences of greatest fibrosis, thickening of the pleura and cavitation. (Fig. 397.)

In patients with chronic tuberculosis, who have been coughing for years, the trachea in addition is frequently dilated, especially in that portion situated just below the cricoid cartilage.

A displacement of the trachea by traction may be found in any disease, aside from tuberculosis, in which there is an induration and fibrosis of the lung. We thus encounter it in chronic pleuro-pulmonary disease of a non-tuberculous nature (fig. 187), in certain forms of bronchial carcinoma and in the indurative form of syphilis of the lung. The displacement may be so extreme that the trachea is drawn completely over into the affected half of the thorax. This is well seen in figure 199 which illustrates the effect of an extensive syphilitic fibrosis of the lung and pleura in a tabetic woman. The adhesions resulting from a chronic pleurisy may also cause a marked malposition of the trachea without any associated fibrosis of the lung. (Fig. 305.)

It is worthy of mention that an extreme displacement of the trachea and mediastinum may be caused by stenosis of a main bronchus with consequent atelectasis of the lung. Here the trachea together with the mediastinum yields to the pressure of the opposite lung, which becomes overdistended. This was apparently the cause of the tracheal displacement shown in figure 19 in which a fibroma so completely occluded the left bronchus, that no evidence of entrance of air into the left lung was found on auscultation. After the removal of the tumor, the left lung promptly expanded and the trachea resumed its central position.

A general displacement of the trachea and mediastinum, without tortuosity, is found in cases of pleural effusion. It is however, not an invariable finding and it depends on different factors, all of which are not entirely clear. The most marked displacement accompanies non-inflammatory effusions which completely fill one side of the chest. On the other hand, in cases of inflammatory exudate, whether free or encapsulated, a displacement of the trachea and mediastinum is frequently absent or very slight and may bear no relation to the size of the effusion. In some cases this may be explained by the presence of encapsulating bands, in others by a fixation of the mediastinum or by a diminution in the volume of the lung on the affected side.

An extreme displacement, perhaps the greatest, occurs in association with pneumothorax with or without effusion. In these patients the trachea may occupy a position beyond the mid-clavicular line of the opposite side. This is particularly noted in cases of valve pneumothorax with a constantly increasing intra-pleural pressure. (Fig. 238.) Tumors of the lung and pleura which are of considerable size usually push the trachea to the opposite side. This is especially true of massive new growths of the upper lobes. (Fig. 207.) With new growths of the pleura the pressure on the trachea is usually due, in part at least, to the associated pleural effusion. Yet, as is illustrated in figure 299, this effect may be produced by the growth alone, without a complicating effusion. A contrary effect may in rare cases be exerted by tumors of the lung or pleura, the trachea and mediastinum being drawn over to the affected side. This phenomenon is seen in figure 208, a case of bronchial carcinoma of considerable size. The mechanism of such an unusual displacement is not clear. Although it may be the result of a scirrhous growth, it must be kept in mind that an occluding carcinoma in the lumen of one or more large bronchi may produce an atelectasis and collapse of the lung with a resulting pressure on the mediastinum from the distended healthy lung.

As may be expected, a trachea which is displaced laterally, so that it comes to lie within the pulmonary fields, may be the cause of abnormal physical signs whose interpretation is fraught with difficulties. When

the displaced trachea is surrounded by infiltrated lung, as in pulmonary tuberculosis, the respiratory sounds along the border of the upper sternum may acquire an amphoric quality identical with that of a cavity. For this reason, the clinician learns to appraise the physical signs of a cavity in this region critically and to palpate the trachea at the root of the neck in order to determine whether it deviates from the mid-line. In cases of extensive fibrosis of the lung, as illustrated in figure 199, the trachea may even be situated in the infra-clavicular region, where physical signs indistinguishable from those of a large cavity may be found.

COMPRESSION OF THE TRACHEA

The commonest site of compression of the trachea is at the root of the neck because here it is confined in the narrowest portion of the thorax and is surrounded by unyielding bone. The compressed trachea is often at the same time displaced to one side or the other. Among frequent causes of a narrowing of the trachea are mediastinal new growths, especially tumors of the thyroid, benign or malignant. Enlargement of a normally situated thyroid usually does not result in compression of the trachea; exceptions to this rule are malignant tumors which actually invade its lumen, or occasionally the tense cysts of a colloid goitre. Thus the trachea may be compressed between an enlarged lobe of the thyroid and a cystic isthmus. A much more common cause of tracheal narrowing is a substernal or intra-thoracic goitre. The narrowing is usually confined to a small area and may be extreme. Stenosis of the trachea from this cause results as readily from adenomatous or colloid goitres as from truly malignant ones.

The various mediastinal neoplasms, especially the lympho-sarcomata and lymphomata including Hodgkin's disease are very prone to produce a compression and deformity of the trachea and the relation of these changes to the most distressing symptoms of these diseases is susceptible of accurate demonstration on the Roentgen plate. In young children tracheal compression finds its most frequent cause in the pressure of tuberculous lymph nodes and rarely in an enlargement of the thymus. Especial attention should be directed to a tumefaction of the para-tracheal lymph nodes on the right side, which may attain a considerable size when they become tuberculous. Because of their position at the side of the trachea, which in infants is easily compressible, marked dyspnoea and stridor may result. In adults, owing to a greater rigidity of the trachea, tuberculous nodes are less prone to produce compression.

Finally, aneurysm of the aorta, from its position in relation to the trachea is calculated to compress it. It is probable that few aneurysms attain any considerable size without diminishing the caliber of the

trachea. The Roentgen plate thus discloses in most cases a distinct decrease in its lumen, due to direct pressure of a sacculation or it may be flattened between two adjacent sacculations. The narrowing of the trachea may however be obscured by the shadow of the overlying aneurysm so that it may be visible only in the oblique position. (Fig. 360.)

TUMORS OF THE TRACHEA

The very rare tracheal tumors are difficult of demonstration because of the obscuring shadows cast by the vertebrae and aorta. In the oblique position however, the lumen of the trachea may be projected into the lung fields, which will supply the contrast necessary to set off a soft tumor within it. Thus in figure 13 is shown a sessile



FIGURE 13

Tumor growing from posterior wall of trachea and projecting into its lumen. Just below it is the bifurcation. Oblique view.

tumor springing from its posterior wall, which was invisible on the antero-posterior plate. For some months this patient had dyspnoea, stridor and recurrent laryngeal palsy. Tracheoscopic examination revealed a carcinoma in the situation indicated on the plate. More often, the trachea is invaded by tumors originating in nearby structures such as the thyroid and the oesophagus. In the case of oesophageal tumors, abnormal shadows are usually not visible and we are led to suspect an involvement of the trachea by the unusual train of symp-



FIGURE 14

Tracheo-oesophageal fistula secondary to carcinoma of oesophagus. Arrow indicates site of tumor. From this point barium passed into the trachea outlining the right lower bronchial tree.

toms which result from a tracheo-oesophageal fistula. Suspicion of this complication is aroused when the ingestion of food is followed by violent, paroxysmal coughing. If we visualize the oesophagus by the ingestion of barium, it is at times possible to observe the passage of the latter through the fistulous opening in the oesophagus into the trachea and finally into the bronchial tree, which may be outlined by the barium to its smaller branches. Thus, in figure 14, the right lower lobe bronchi are full of barium, radiating from the funnel-shaped stenosis of the oesophagus.

The ingestion of food may also be followed by violent coughing whenever the pharyngeal muscles are paralyzed, notably in cases of bulbar palsy. As in cases of tracheo-oesophageal fistula, the bronchial tree may be outlined by the ingested barium. There is this difference



FIGURE 15

Bronchi filled with aspirated barium in case of bulbar palsy. Trachea also outlined by barium.

however; the barium will also be visible in the upper trachea and examination of the oesophagus will fail to disclose an obstruction to the downward passage of the meal. Thus, in the case illustrated in figure 15 during the fluoroscopic examination the barium passed directly into the larynx and then downward into the bronchi. One day later the patient died of respiratory failure due to bulbar palsy.

CHAPTER IV

The Bronchi

Although the bronchi are not distinguishable on the Roentgen plate from the other structures which go to form the lung markings, we can nevertheless by a correlation of clinical and Roentgen data, derive considerable information regarding bronchial diseases.

CHRONIC BRONCHITIS

Roentgen examination of the chest is probably undertaken more often for the diagnosis of chronic bronchitis or its underlying causes than for any other disease. When we interpret the Roentgen plate in this disease it is important to realize that the abnormal shadows which may traverse the lungs are only to a minor degree the result of inflammatory changes in the bronchi. They are to a greater degree the result of associated conditions such as pulmonary emphysema and a general fibrotic process which may affect not only the peribronchial but also the peri-vascular and interstitial connective tissues. In long standing cases we therefore find the lung markings abnormally prominent, this being due both to an increase in the connective tissue of the lung and to the greater contrast furnished by the overoerated lungs. The latter are usually emphysematous and are contained within a bony thorax which has acquired the well-known barrel shape of emphysema and chronic bronchitis. In addition, the bronchial nodes which are chronically inflamed, are enlarged and indurated so that the hilum extends into the lung fields for some distance. The low, flat diaphragm which scarcely moves in respiration, the median, narrow heart and the calcified costal cartilages complete a rather characteristic Roentgen picture.

However, the value of the Roentgen examination consists less in the demonstration of these changes than in the discovery of abnormal conditions in the heart and lungs, to which the bronchial disease may be secondary. The thoughtful clinician is not content with a diagnosis of chronic bronchitis until he has excluded other more serious disease as a cause of it. It is frequently only a symptom of a more grave affection which, because of the associated emphysema may be completely masked. It therefore frequently happens that the Roentgen plate brings to light an apical indurative tuberculosis, the physical evidences of which are obscured by the bronchitis. In fact, the so-called asthmatic form of tuberculosis in older individuals, may clinically furnish no other evidence of its existence than a chronic bronchitis and asthma and only the Roentgen examination may reveal the underlying tuberculous process.

Similarly, a new growth of the lungs, chronic cardiac disease and the cardiac hypertrophy of chronic nephritis may be discovered as the underlying causes of a chronic bronchitis and decisive light may thus

be thrown on an otherwise obscure case. Such experiences invest the diagnosis of chronic bronchitis with so much uncertainty that the clinician hesitates to make it until he has exhausted the resources of his examination.

BRONCHIECTASIS

In the clinical differentiation of the various types of bronchial dilatation, it is of value to distinguish between the multiple, usually small dilatations which result from a gradual weakening of the bronchial wall and the gangrenous bronchiectases which are such an assential element in the cases of acute and sub-acute pulmonary sup-puration and gangrene. A discussion of the latter we will reserve for the chapter on Abscess and Gangrene of the lung.

Bronchiectases are usually the sequel of a pneumonic process in which the growth of fibrous tissue about the small bronchi results in their gradual dilatation, probably under the influence of a prolonged period of coughing. In interpreting the Roentgen plate in this disease, we have therefore to keep in mind the existence of two pathological changes, one, the pneumonic infiltration, the other, the bronchial dilata-tion. The Roentgen appearance in different cases may be quite dis-similar depending on the relative intensity of these two processes. Thus, multiple small bronchial dilatations lying within a consolidated lung will not be visible through the dense shadow of the lung about them. The Roentgen shadow will therefore be simply that of a pneu-monia. On the other hand, when for any reason the bronchiectases attain a larger size and become globular, they may contain so much air that they are visible even through the shadow of an indurated lung.

We may roughly distinguish two forms of multiple bronchiectases on the Roentgen plate. In the less common type one or more lobes of the lung are occupied by numerous globular cavities whose walls are formed by the thinned out, dilated bronchioles. The cavities are often of considerable size, their diameter being at times as much as one inch. Lying next to each other and entirely replacing the lung parenchyma, they may convert the lung into a honey-comb or sponge-like structure. (Fig. 16.) Usually, this form of the disease is acquired in childhood subsequent to a pneumonia, which is followed by a long period of cough-ing during which the characteristic symptoms, consisting among others of profuse purulent expectoration and clubbing of the fingers, develop. This appears to represent a mild form of the disease, which is borne with very little discomfort and is consistent with robust health for many years. The probable reason for this becomes evident when we study the Roentgen plate in these cases. It will be noted that there is no evidence of a pneumonic process which, as we shall see, is a potent factor in extending and perpetuating the bronchiectatic process. It is probable that here the pneumonic process has completely subsided, per-

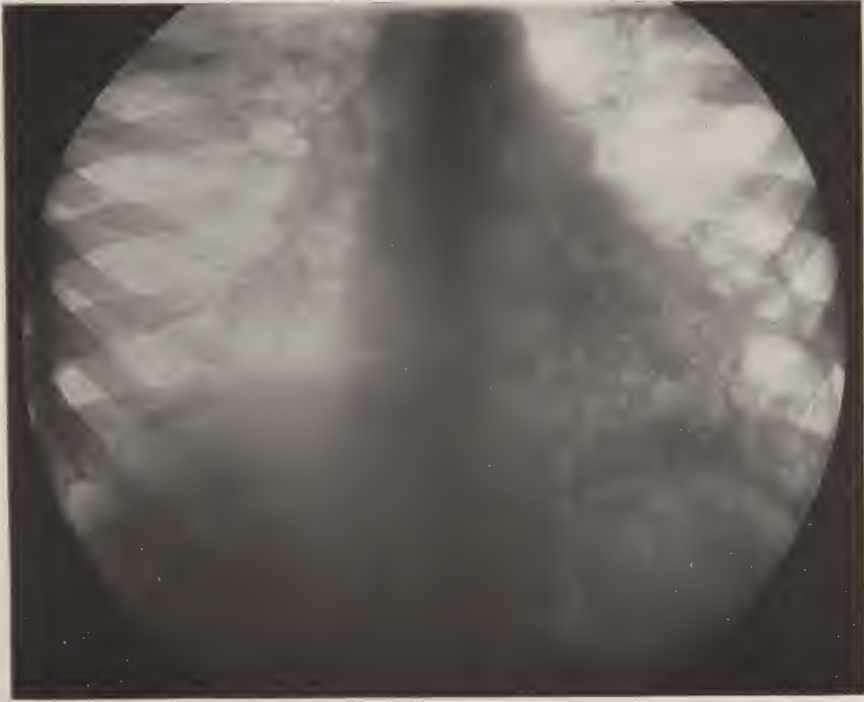


FIGURE 16

Multiple bronchiectases, showing typical spongy appearance of left lower lobe; also at root of right lung.

haps years ago and that the disease is now a purely bronchial one. The patients therefore suffer from the mechanical effects of a bronchial dilatation, with a super-added bronchitis and a stasis of accumulated bronchial secretion. Aside from the discomfort incident to the evacuation of this secretion, they may enjoy good health for many years. The physical signs of this affection are rarely definite enough to warrant a diagnosis of bronchiectasis, which is rather made from the history, the symptoms and the almost constant presence of clubbed fingers. The cavities are too small to furnish characteristic signs, which are only those of a bronchitis. In some patients the bronchitis may even subside to such a degree that the sputum is no longer profuse; under these circumstances, the condition may pass entirely unrecognized by the physician. For example, the middle aged patient, whose left lung presented the numerous bronchiectases seen in figure 17, had only a slight morning cough for many years and passed through a major operation under general anesthesia with no untoward results. In these cases the physician is entirely dependent on the Roentgen examination for a diagnosis.

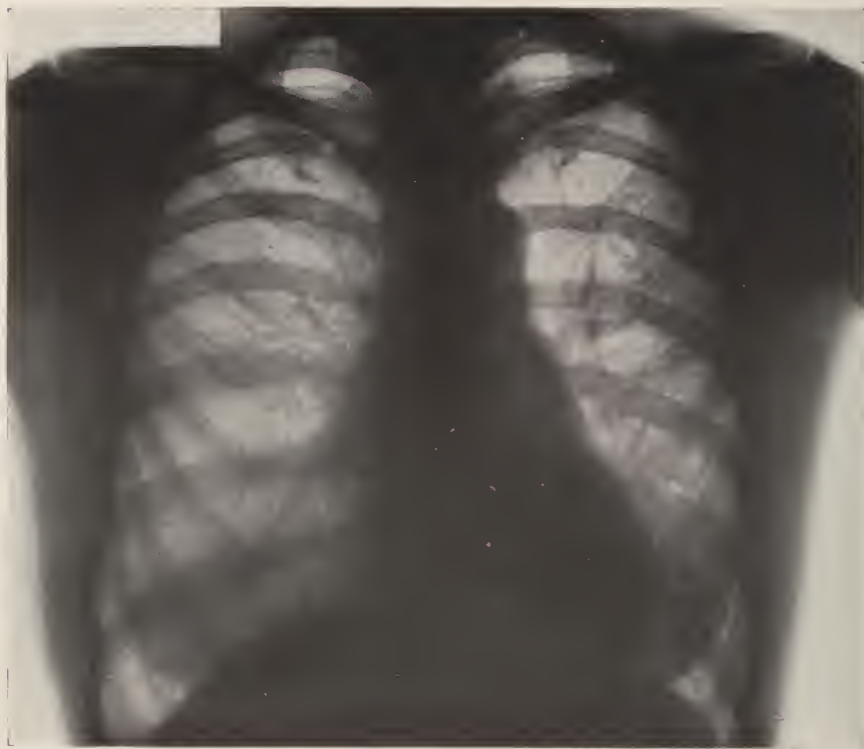


FIGURE 17

Multiple globular dilatations of bronchi in left lower lobe.

In the more frequent form of bronchial dilatation, the pneumonic process to which it is secondary is a persistent one and leads to greater fibrosis which in turn tends to aggravate the bronchiectases. In general, the prognosis in this form of the disease must be regarded as graver than in the previous one as it is often later complicated by pulmonary suppuration and gangrene. It therefore not infrequently happens that a patient will pass through an acute pneumonia, which persists and organizes, followed by a period of subacute pneumonitis during which bronchiectases are formed. Following a variable interval of months or years, during which large amounts of purulent sputum are brought up, anaerobic infection of the lung may supervene, with the complete picture of lung abscess and gangrene.

As a rule in this group of cases the bronchial dilatations are small and cylindrical in shape, as they are probably prevented from becoming larger and globular by the surrounding indurated lung. They are also obscured by the latter, so that on the Roentgen plate we see only a dense pneumonic shadow, within which occasionally the outlines of

enlarged bronchioles, replete with secretions, are faintly indicated. Rarely, the bronchiectases, being larger and perhaps more superficially situated, are visible even in consolidated lungs. (Fig. 18.) In this



FIGURE 18

Multiple bronchiectases within a chronically indurated lung.

patient, a child of eight years, the disease was of four years duration and followed pneumonia. The characteristic symptoms of bronchial dilatation were present and in addition, at various times, the sputum was fetid, an almost certain indication of an actual involvement of the lung in a gangrenous process.

It will be evident that the Roentgenologist must be prepared to make the diagnosis of multiple bronchiectases on meagre data. Often he sees no evidence of them on the plate and he must infer their existence from the presence of a pneumonic shadow. This, however, he can only safely do if he has the support of the clinical history and physical findings.

The influence of disease of the pleura on the development of bronchiectases must also be considered. The fibrosis which may begin on the surface of the lung secondary to an empyema, may penetrate the parenchyma and give rise to so-called pleuro-pneumonic bronchiectases. These cases are difficult to interpret by the Roentgen examination alone because the fibrosis may cause such a thickening of the pleura as to obscure the more deeply seated disease. In cases of unoperated or unrecognized empyema, a slowly progressive inflammatory process may lead in a few months to the formation of bronchial dilatations, the striking symptoms of which may completely divert attention from the untreated empyema; the first sign of the latter may be its rupture into a bronchus and the consequent expectoration of a large amount of pus. Here a careful clinical history is of the greatest importance in order to determine the sequence of events because the converse possibility exists of primary bronchiectases with rupture of one of them into the pleural cavity resulting in an empyema. Instances of the latter are not uncommon and they explain certain cases of persistent empyema in which the ruptured bronchus becomes the starting point for the formation of a pleuro-pulmonary fistula. This sequence of events must especially be kept in mind in the case of children in whom bronchiectases following a pneumonia or the aspiration of a foreign body may be unrecognized until their rupture and the resultant empyema draw attention to them.

The multiple cylindrical or globular dilatations which are a result of the distention of terminal bronchioles must be distinguished from a diffuse enlargement of the larger bronchi. The latter are usually present in lung abscess and gangrene and although not visible on the Roentgen plate, they can be seen through the bronchoscope. There are also rare forms of chronic indurative pneumonia in which the terminal bronchioles are unaffected, the dilatation involving only the larger tubes. However, this widening of the bronchi is only exceptionally visible on the plate as they are completely obscured by the pneumonic lung about them.

STENOSIS OF THE BRONCHI

Of the various causes of stenosis of a main bronchus, certain ones such as an aneurysm or an extra-bronchial tumor may readily be seen on the plate. The mechanical effect on the lung of such a stenosis is often an impairment in the aeration and a consequent atelectasis of the area of the lung supplied by the bronchus. This manifests itself on the plate as a homogeneous cloudiness of the lung which may be of any grade, from a slight haziness to an appearance indistinguishable from a consolidation. When the stenosis is due to a tumor the shadow of the atelectatic lung may merge with that of the tumor so that the latter will appear larger than it actually is. Thus, in figure 210 the shadow

in the left lower lobe has two components. The portion near the root of the lung was cast by a small adeno-carcinoma originating in a main bronchus, which it occluded. The greater part of the shadow was however due to a collapse of the left lower lobe. The physical signs, consisting of dulness and absent respiratory sounds were naturally enough, though erroneously, attributed to a pleural effusion.

A valuable sign of stenosis of a bronchus may be observed on the fluoroscopic screen. This consists in a displacement of the mediastinum toward the affected side during inspiration and is caused by the expansion of the normal lung which is unopposed by that of the atelectatic lung. This phenomenon may be of value in the diagnosis of obstruction by bands in bronchial syphilis, by endobronchial growths, by bronchial

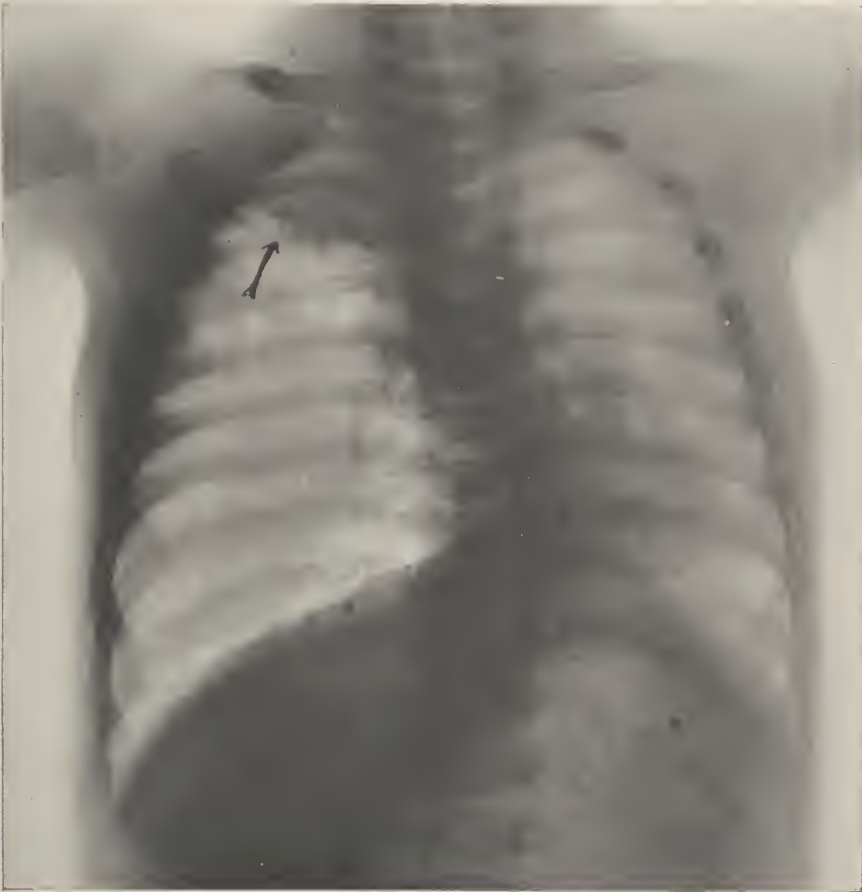


FIGURE 18a

Broncho-stenosis by tuberculous nodes. Atelectasis and collapse of right upper lobe. Partial occlusion of lower lobe bronchus with over-distension of lobe, displacement of mediastinum to left and depression of diaphragm. Arrow points to compressed, airless upper lobe.

exudates as in diphtheria or by foreign bodies which cast no shadows on the plate. It is not however, exclusively found in these conditions. In any pulmonary disease in which the alveolar area is much diminished, as by extensive fibrosis, a similar movement of the mediastinum may be seen.

Under special conditions, when a bronchus is only partially obstructed, there may result an over-distention of the affected lobe rather than an atelectasis. Such a phenomenon is occasionally observed with foreign bodies which partially occlude a bronchus in such a way that air may more easily enter the lung than it can be expelled. The lung therefore becomes progressively more and more distended to such an extent that the mediastinum may be displaced and the diaphragm depressed. It is probable that edema of the mucous membrane at the site of the obstruction contributes to this valve-like action. In figure 18a are illustrated in the same case the effects of a complete obstruction of one bronchus and the partial obstruction of another by a mass of tuberculous nodes in the superior mediastinum. It will be noted that the right upper lobe is completely airless so that it has contracted to only a fraction of its normal size, its lower border being at the level of the second rib. On the other hand there is a partial occlusion of the right lower bronchus as a result of which the lower lobe has become over-distended. It has crowded the upper lobe to the apex of the chest, it extends far beyond the mid-line to the left side and the diaphragm is depressed. These changes were discovered in a child of three months who presented paroxysmal attacks of dyspnoea and a more or less constant stridor. Tuberculin test positive.

TUMORS OF THE BRONCHI

The malignant tumors which take their origin in the large bronchi rapidly invade the adjacent lung; their diagnosis may therefore more logically be considered in the chapter devoted to pulmonary new growths. When such tumors are still confined to the bronchus, they cannot be diagnosed as such by a Roentgen examination.

Benign tumors which spring from the wall of a large bronchus will also elude the examiner; the changes which may result from occlusion of the bronchus such as atelectasis of the lung or secondary suppuration, present nothing on the plate that is characteristic. Only the physical signs, such as a complete absence of breath sounds or symptoms suggesting the obstruction of a bronchus, may arouse a suspicion of the real condition. The great extent of the secondary changes in the lung from even a small endo-bronchial tumor are illustrated in figure 19.



FIGURE 19

Fibroma of left bronchus. Stenosis of bronchus with atelectasis of lung. Trachea and heart displaced to left.

This patient for a few months suffered with a dry harrassing cough and attacks of dyspnoea, to which was later added a profuse expectoration of purulent sputum. Physical examination revealed a marked decrease in expansion of the left lung, together with a complete absence of breath sounds on that side. There was no fluid in the left chest. The plate shows a retraction of the left chest, displacement of the mediastinum and heart to the left by the emphysematous, uninvolved lung and a replacement of the left lung field by a dense shadow. The signs all pointed to an obstruction of the left bronchus. This was corroborated by the bronchoscopic examination which disclosed a fibroma occluding the left main bronchus. The rapid improvement which followed the removal of this tumor is remarkable. Within two weeks, hand in hand with the expectoration of much retained mucus and pus, the left lung became completely aerated and the heart returned to its normal position. It is probable that in addition to an atelectasis, there was a pneumonic process from retained secretions, which had not yet developed a permanent character and was still capable of resolution.

Long Rocks for
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SECTION IV

The Lungs

Pulmonary Tuberculosis in Adults.

Pulmonary Tuberculosis in Children.

Pneumonia.

Pulmonary Suppuration: Abscess and Gangrene of the Lung.

Chronic Non-tuberculosis Infections:

Indurative Pneumonia,

Actinomycosis,

Echinococcus Cyst,

Syphilis.

Tumors of the Lungs:

Primary Tumors,

Metastatic Tumors.

CHAPTER V

Pulmonary Tuberculosis in Adults

There are few diseases whose prompt and accurate diagnosis is of greater moment to the patient, than pulmonary tuberculosis. It is therefore gratifying that in the Roentgen Ray we possess a method of examination which in its accuracy and timeliness is rivalled by few diagnostic procedures.

It must however be remembered that like any diagnostic method its value will be measured by the technical skill of the physician in the making of plates no less than his ability to interpret them. The plate must be clear and its markings well defined in order that the normal shadows may be distinguished from the pathological. The interpretation of hazy or otherwise imperfect plates has as little justification as a physical examination of the lungs without disrobing the patient.

Skill in interpretation presupposes not only a familiarity with Roentgen shadows but also a working knowledge of the clinical and pathological features of tuberculosis. Unless such knowledge is at the disposal of the examiner he will not be in a position to profit fully from his study of the plate nor will he be able to express in appropriate language, as is often possible, the clinical or pathological type of the disease. It is scarcely necessary to point out that only a physician is possessed of the knowledge which is a necessary pre-requisite of such an intelligent interpretation. It is an affront to the clinician to offer him, as is so often done, the opinion of a non-medical technician in a case of tuberculosis. The Roentgen diagnosis will inspire respect in the mind of the clinician commensurate with the medical knowledge of the Roentgenologist.

METHODS OF EXAMINATION

There are two methods which may be employed in the Roentgen diagnosis of pulmonary tuberculosis, fluoroscopy and radiography. Although in the early days of Roentgenology these methods enlisted opposing adherents, it is now agreed that they are co-ordinate parts of a complete examination. There was a time when, for technical reasons, good chest plates were difficult to make, so that there may have been grounds for preferring the fluoroscopic screen. Individual workers by dint of practice became expert in the interpretation of screen findings, yet they became so, not from choice but from necessity because good chest plates were not available to them. Fluoroscopy can reveal only the coarser shadows associated with more advanced disease of the lung; it often fails to discover the finer infiltrations of incipient tuberculosis.

The fluoroscope nevertheless has a definite and unique function in the Roentgen examination. It should always precede the making of the plate and any data thus ascertained should be utilized to supplement the radiographic findings. It is of supreme value in the observation of moving structures such as the diaphragm and the heart and of the shifting fluid levels in cavities and in the pleura. It is often of greater value than the plate in determining the extent and localization of areas of pneumothorax and of effusions and it has the advantage of a view of the chest in any desired position of the patient. Slight differences in aeration are perceived more easily on the screen and this is particularly true of the apices, where atelectasis is so often found.

Having conceded these advantages of the screen, it must be repeated that the ultimate diagnosis, especially of incipient tuberculosis, must be based on a well executed Roentgenographic examination. It is especially unsafe to deny the existence of tuberculosis of the lung from negative fluoroscopic findings.

STEREOSCOPIC VERSUS SINGLE PLATES

Stereoscopic examination of the chest affords us a plastic image of the lungs in which the ramifications of the pulmonary vessels are visible with remarkable clearness. Infiltrations whose depth and density can only be guessed at on the single plate are seen in their true relation to surrounding structures and their proximity to the anterior or posterior chest wall can be determined. This applies particularly to deposits on the pleura, the shadows of which cannot otherwise be distinguished from pulmonary infiltrations with any degree of certainty.

So much can fairly be said for the stereoscopic examination. There is, however, a tendency among Roentgenologists to exaggerate its importance, to consider it indispensable for the diagnosis of pulmonary tuberculosis and finally to regard a single plate as of minor value. Such a view we wish to controvert as emphatically as we can because it is contrary to fact. True, to those who have not mastered the art of making excellent single plates the striking plasticity and apparent detail of even a mediocre stereoscopic set, will be pleasing to the eye. Yet, if the truth were told, in many of these cases the Roentgenologist has achieved nothing more than a pleasing optical illusion.

The test of a good Roentgen examination is the single plate. If it is properly made, so that its markings are sharply defined and free from haziness and distortion, the examiner will have no difficulty in distinguishing the infiltrations of tuberculosis. On the other hand, if a proper technique is not observed, stereoscopy will avail him as little as the single plate.

INCIPIENT TUBERCULOSIS

The earliest lesions of tuberculosis are regularly found somewhere in the upper lobes, usually near the apex. They are nearly always unilateral. On the plate they appear as distinct circumscribed shadows which may be solitary or as is oftener the case they occur in groups of smaller shadows with indistinct outlines. The individual shadows may be as small as millet seeds or they may be one to several centimeters in diameter. Usually they are roughly circular although they may exhibit a great variety of shapes, which are probably dependent on associated inflammatory changes.

A common type of beginning tuberculosis is illustrated in figure 20 and figures 21 and 21a. The deposits are fairly large and well outlined,



FIGURE 20

Early tuberculous process in left infraclavicular region. (Arrow.)



FIGURE 21

Early tuberculous infiltration in left infraclavicular region. (Arrow.)

although of only moderate intensity, so that they are easily obscured by overlying ribs and muscles. Again in figure 22 an exceedingly faint, almost circular infiltration is distinctly seen as it is projected on the first



FIGURE 21a

Left apex of figure 21, natural size, showing infiltration of early tuberculosis in infraclavicular region. (Arrow.)



FIGURE 22

Early tuberculous lesion below right clavicle.

right intercostal space. In figure 23 may be seen an early involvement of the very summit of the right apex.



FIGURE 23

Early tuberculosis at extreme apex of right lung.

The smaller infiltrations, probably composed of miliary tubercles, may be grouped into masses of tiny shadows of different densities, (figs. 24 and 25 and 24a and 25a) which lack the homogeneous appear-



FIGURE 24

Early tuberculosis at right apex. Large group of tubercles surrounded by pneumonic exudate.



FIGURE 24a

Right apex of figure 24, natural size, showing early tuberculous infiltrations in infraclavicular region.



FIGURE 25

Early tuberculous process below left clavicle. A group of tubercles imbedded in pneumonic exudate.



FIGURE 25a

Left apex of figure 25, showing a group of small infiltrations of beginning tuberculosis in the infraclavicular region.

ance of the solitary deposits. Such miliary tubercles cannot of course be recognized as infiltrations unless they occur in groups, which is usually the case. In figure 26 both apices have a faintly granular appearance from large numbers of closely studded tubercles.



FIGURE 26

Bilateral apical tuberculosis, consisting of numerous tubercles.

Of course, these early infiltrations may also be seen in cases of more advanced tuberculosis when for any reason there is a renewal of the infection. Their great variability in size and shape is well illustrated in figure 27 where they are associated with the more advanced lesions shortly to be described.

In picturing to ourselves the pathological changes which are responsible for these diverse shadows in beginning apical disease, two facts must be borne in mind. First, in incipient disease, before necrosis of the tuberculous tissue has taken place, the infiltrations have little absorptive power for the Roentgen Rays. The shadows are therefore



FIGURE 27

Illustrates a great variety of tuberculous infiltrations. In left infraclavicular region, the deposits have the faint shadows of recent lesions. On right side the infiltrations are undergoing fibrosis.

faint and lack the intensity which, as we shall see later, is a consequence of caseation. Secondly, an almost invariable accompaniment of the tubercle is a zone of exudative inflammation in the alveoli about it. This accounts for the indistinct outlines of many of the earliest deposits and the homogeneous shadows which they may cast. These features of early tubercles are seen in figure 24 where there is only a very faint cloud-like shadow in the right infraclavicular region, in which the tubercles are obscured by the surrounding exudate.

In the further course of our study we shall see that these two pathological processes, caseation and pneumonia, together with the subsequently developing fibrosis, will determine to a great extent the Roentgen appearance of tuberculosis of the lungs in its protean manifestations.

AT HOW EARLY A STAGE CAN TUBERCULOSIS BE DIAGNOSED?

It is apparent, from the facts thus far presented that relatively small deposits at an apex are susceptible of certain demonstration on the Roentgen plate. It is proper to inquire at this point whether the shadows we have described represent the actual beginnings of the disease or whether there has preceded a stage in which the infiltrations were too small to cast recognizable shadows. Although this query does not admit of a positive answer, experiment has thrown some light on it. It has been determined that an area of infiltration, situated near the surface of the lung must be at least 4 mm. in diameter in order to cast a distinctive shadow on the plate. The more deeply seated the diseased area, the less clearly will it be perceived.

Obviously, a solitary miliary tubercle, assuming that it may usher in the disease, cannot be recognized as such on the plate; if it casts a shadow at all it will not be distinguishable from the shadows of bifurcating or intersecting blood vessels which contribute to the normal mottling of the lung. It may be taken for granted therefore that it is possible for a tuberculous focus to exist, which is so small as to escape discovery by the Roentgen Ray. Tuberculosis of the lungs offers no exception to the rule that diseases are never discovered in their very incipency by the clinical means at our disposal. A latent period intervenes during which the foci of the disease increase in size and activity until they make themselves clinically evident.

It is questionable, however, whether in actual practice this circumstance seriously affects the value of the Roentgen examination. In the first place, as we have stated, the earliest deposit of tubercles is frequently associated with a surrounding pneumonia which measurably increases the area of infiltration. A more practical consideration is the fact that during its incubation the symptoms of the disease are apt to pass unrecognized by the patient and for this reason, the question of diagnosis, whether clinical or Roentgenological, does not arise. It is our experience that when pulmonary tuberculosis is of sufficient extent to produce clinical symptoms, it is already susceptible of Roentgen demonstration.

THE LOCATION OF THE EARLIEST LESIONS

The Roentgen Ray has disclosed the interesting fact that the earliest evidences of the disease are less often found in what is clinically known as the apex of the lung than in the infraclavicular region. Furthermore, the axillary half of the lung is more apt to be involved in the beginning than the mesial half. (Fig. 28.)



FIGURE 28

Incipient tuberculosis, right infraclavicular region. This is the earliest recognizable type of tuberculosis. This is also the most frequent site for the beginning of the disease.

The bearing of these facts on the clinical diagnosis of incipient lesions is an important one as it throws some light on the discrepancies so often found between the physical and the Roentgen examination. In looking for rales or breathing changes the clinician customarily devotes most attention to the extreme apex of the lung. If perchance the tuberculous process is situated at the apex, he will be successful in his search. On the other hand, in a large percentage of early cases, the infiltrations occupy exclusively the infraclavicular region and abnormal physical signs are accordingly absent at the apex. But they are also frequently absent below the clavicle because the transmission of adventitious sounds is hindered by the pectoral muscles. It therefore happens that the large group of cases in which the infiltrations first make their appearance in the infraclavicular region are unrecognized by the clinician for some time. In these patients physical signs may come to light only later when the process has extended to the apex. In other words, a process which may clinically involve only the extreme apex and therefore seem to be incipient, is often found by the Roentgenologist to involve also the infraclavicular region and therefore to be moderately advanced.

Unlike the physical examination, the Roentgen plate enjoys a distinct advantage in the diagnosis of infraclavicular lesions. In this region, far removed from the complicated shadows at the root of the

lung, the pulmonary fields have an almost homogeneous texture, as the arborizations of the small vessels are almost invisible. Infiltrations in the lung can therefore easily be recognized as they stand out against the background of aerated lung. This is not true to the same degree of shadows in the mesial half of the lungs. The presence near the pulmonary hilum of the larger blood vessels and of masses of lymphoid tissue which may be enlarged by disease, may render a distinction between the normal markings and pathologic deposits difficult. For this reason, the Roentgenologist will have to exercise caution in the interpretation of the nodular shadows which he will frequently see in the mesial half of the lungs. If there is no evidence of tuberculosis at the sites of selection which we have indicated, he will wisely err on the side of caution and regard the questionable shadows as due to distended blood vessels and lymphatics. Although a rule of this sort may be considered arbitrary, it is sanctioned by experience which teaches that isolated tuberculosis of the mesial part of the lung is uncommon, whereas involvement in other locations is the rule.

In very rare cases the first evidence of tuberculosis may be found in the lower part of the upper lobe or even in the lower lobe. We shall see in a subsequent discussion that this unusual site of the disease is not uncommon in infants and in children. In adults, it is undoubtedly among the rarities and clinical investigation of such cases will often bring to light some underlying local or systematic condition which has favored the unusual location of the tuberculous process.

IS THE ROENTGEN APPEARANCE OF TUBERCULOSIS TYPICAL OF THIS DISEASE?

The Roentgenologist is often asked whether the abnormal shadows which we have described are characteristic of tuberculosis and whether, based on them alone, we are warranted in diagnosing this disease. May not such shadows be cast by pathological processes other than tuberculosis?

Strictly speaking, a single shadow or a group of shadows has no intrinsic tuberculous character. Any inflammatory deposit or even a neoplasm, may cast shadows which occasionally simulate those of tuberculosis. In exercising his judgment therefore, the Roentgenologist must take into account such facts in the clinical history of the case and in the pathology of lung diseases which will help him to exclude causes for the abnormal shadows other than tuberculosis. We may accept it as a cardinal rule however, that infiltrations in the apical and infra-clavicular regions are tuberculous, especially if they are small, nodular, and occur in groups. The exceptions to this rule are few and, in practice, unimportant. For example, occasionally a resolving pneumonia of an upper lobe may leave a residuum of infiltration at the apex which, in

the size and arrangement of its shadows may resemble an early tuberculous process. The clinical symptoms may or may not be of help in distinguishing between the two conditions. Obviously, the ultimate decision may have to wait on a subsequent examination after complete resolution, which will involve a delay of several weeks, at the most.

Again, an early metastatic new-growth, especially when it is solitary, may closely resemble an incipient tuberculosis or a tuberculous infiltration may simulate a new growth. (Fig. 29.) Error can usually



FIGURE 29

Circular infiltrations of tuberculosis resembling metastatic tumors.

be avoided by the clinical examination which will discover a primary growth and also by a later increase in the size and number of the metastases. In these cases the Roentgen examination should be repeated after a suitable interval in order to determine a significant change in the shadows. It is scarcely necessary to point out that in doubtful cases the Roentgenologist should no more be denied the opportunity to check up his findings, than the clinician.

ASSOCIATED CHANGES IN THE CHEST IN INCIPIENT TUBERCULOSIS

A complete Roentgen examination in cases of suspected tuberculosis should always include a preliminary fluoroscopy. Although the screen findings in incipient cases are rarely decisive by themselves, they may be suggestive and in the case of doubtful plates, they may be corroborative.

In the first place, fluoroscopy is better adapted for the determination of a slight deficiency in the aeration of an apex which is a common accompaniment of early lesions. In the interpretation of such a deficiency, the various causes for it must be kept in mind. The air content of an apex may be diminished and the apex may therefore, appear less illuminated because of actual infiltration which reduces the amount of aerated lung. In cases of this type the apex remains darker even after deep breathing and after coughing. Such a persistent atelectasis is therefore strongly suggestive of actual disease of an apex, a presumption which will often be corroborated by a positive plate finding. It should however be remembered that the same phenomenon may be observed in the case of healed lesions at an apex with thickening of the pleura or with fibrosis and retraction. As such healed lesions are very common in otherwise healthy individuals, the value of a unilateral diminution in aeration is very much restricted. An atelectasis may moreover have other causes than actual infiltration. A small deposit at an apex, by reflex inhibition may restrict the movement of the upper chest and therefore interfere with its ventilation. In these cases, the lung can be caused to clear up by deep breathing or coughing.

A slight diminution in the aeration of an apex can only be gauged by a comparison with the other side. In case both sides appear darker than the normal, there is no longer a standard for comparison and any judgment as to aeration should be made with caution. There are numerous causes, aside from tuberculosis, which may obscure the apices. Some of these have no relation to their air content, such as fat deposits in the supra-clavicular region or excessive muscular development and an unusual conformation of the bony thorax. Cervical ribs, calcification of costal cartilages or over-lapping of the sternal ends of the clavicle and the ribs may entirely obscure the apices. Again, some individuals habitually under-aerate their apices; this occurs especially in mouth breathers in whom the so-called collapse atelectasis is found at both apices. It will be seen that the fluoroscopic finding of a diminished aeration, either on one or both sides, has a limited value; ultimately, the Roentgen diagnosis of tuberculosis must be based on a positive plate finding.

A corroborative sign of apical tuberculosis is immobility of the diaphragm on the affected side. This occurs in a large percentage of early cases and in conjunction with a cloudy apex, creates a strong presumption of recent disease. The reason for this immobility is not quite clear. It is evidently not a paresis of the diaphragm because the latter is not found in the expiratory position as in cases of acute pleurisy or pneumonia. The diaphragm appears rather to be in a condition of tonic contraction; it is normal or low in position and the immobility may perhaps be best explained as a reflex rigidity similar to that of the intercostal and pectoral muscles in the vicinity of the apical process.

A number of other abnormalities are to be noted in the chest of cases of early tuberculosis which have no direct bearing on the apical disease itself. It is not uncommon to find evidences at the extreme apices of a previous lesion which has resulted in retraction and scarring and it is important in interpreting the plate, not to assign an undue importance to these changes.

The same observation may be made in regard to the enlarged lymph nodes at the roots of the lungs. A more detailed discussion of these will be reserved for a later chapter. It will suffice, for the moment, to point out that enlarged, calcareous and caseous regional nodes are an almost regular accompaniment of pulmonary tuberculosis. Their size however bears no significant relation to the extent or nature of the infiltrations in the lungs. Tuberculous bronchial lymph nodes are so commonly present in otherwise healthy individuals that their discovery by the Roentgen examination has no clinical significance and they should therefore bear no weight in our estimate of the case.

THE PROCESS OF HEALING IN INCIPIENT TUBERCULOSIS

Tuberculosis of the lungs has an inherent tendency to become quiescent and ultimately to heal. It is natural therefore to inquire into the changes, if any, which take place in the Roentgen shadows of this disease when the process has become arrested and is going on to cure.

If we observe a group of infiltrations which are undergoing involution, the first change noted will be a disappearance of the inflammatory zone about them, which has given the shadow its ill-defined outline. The shadows therefore acquire a sharper, more definite contour. Frequently also, they become denser owing to partial caseation of the tubercles. An illustration of these changes is seen in figure 30. There may further be a progressive decrease in the size of the shadows which ultimately are barely visible. (Fig. 31.) It must be borne in mind that



FIGURE 30

Healing of tuberculosis. On left side, below the clavicle is seen an area of fibrosis which has completely replaced the tuberculous infiltrations. On the right side, the individual tubercles can still be made out. They are however acquiring the sharpness of outline preceding fibrosis.

this transformation is very gradual, requiring usually years before it is completed, during which period the alteration in the shadows is imperceptible. It is probable moreover, that only the smallest incipient lesions are capable of complete disappearance; more extensive infiltrations practically always leave in their wake distinct evidence of their previous existence, which is due to the introduction of two new elements in the healing process, namely, fibrosis and calcification.



FIGURE 31

Healing tuberculosis. Absorption of early infiltrations without fibrosis. The shadows at right apex are exceedingly faint and small.

The development of fibrous tissue offers us the clearest evidence of healing of the disease. We may find it as a fine or coarse network which is woven into the infiltrated area or after the absorption of the latter, it may entirely replace the tuberculous tissue. We may graphically trace the transition from infiltrations to pure fibrosis by observing a series of plates of different individuals in which all the phases of this process are represented. Figures 27, 32 and 33 portray the earliest



FIGURE 32

Healing of early tuberculous infiltration in left upper lobe. The shadows are more sharply defined and elongated, indicating beginning fibrosis.



FIGURE 33

Healing tuberculosis at right apex. Shadows are sharply defined and dense.

stages of fibrosis. The infiltrations lose their distinctness and merge into one another; they are elongated and assume the radial disposition of the lung markings, from which however, they can be readily distinguished. A later stage of the same process is seen in figure 30 where the individual infiltrations below the left clavicle are no longer visible because they lie in a coarse network of fibrosis which is strictly local-



FIGURE 34

Fibrous network of healed tuberculosis in left infraclavicular region. See figure 34a.

ized to a small area. A similar process is beginning to take place on the right side. The complete metamorphosis of the lesion, consisting of its entire replacement by fibrous tissue is well shown in figures 34, 34a, 35 and figure 36. In the last, only a single linear strand of scar



FIGURE 34a

Left apical region of fig. 34, natural size, showing development of fibrosis in a small tuberculous infiltration. (Arrow)

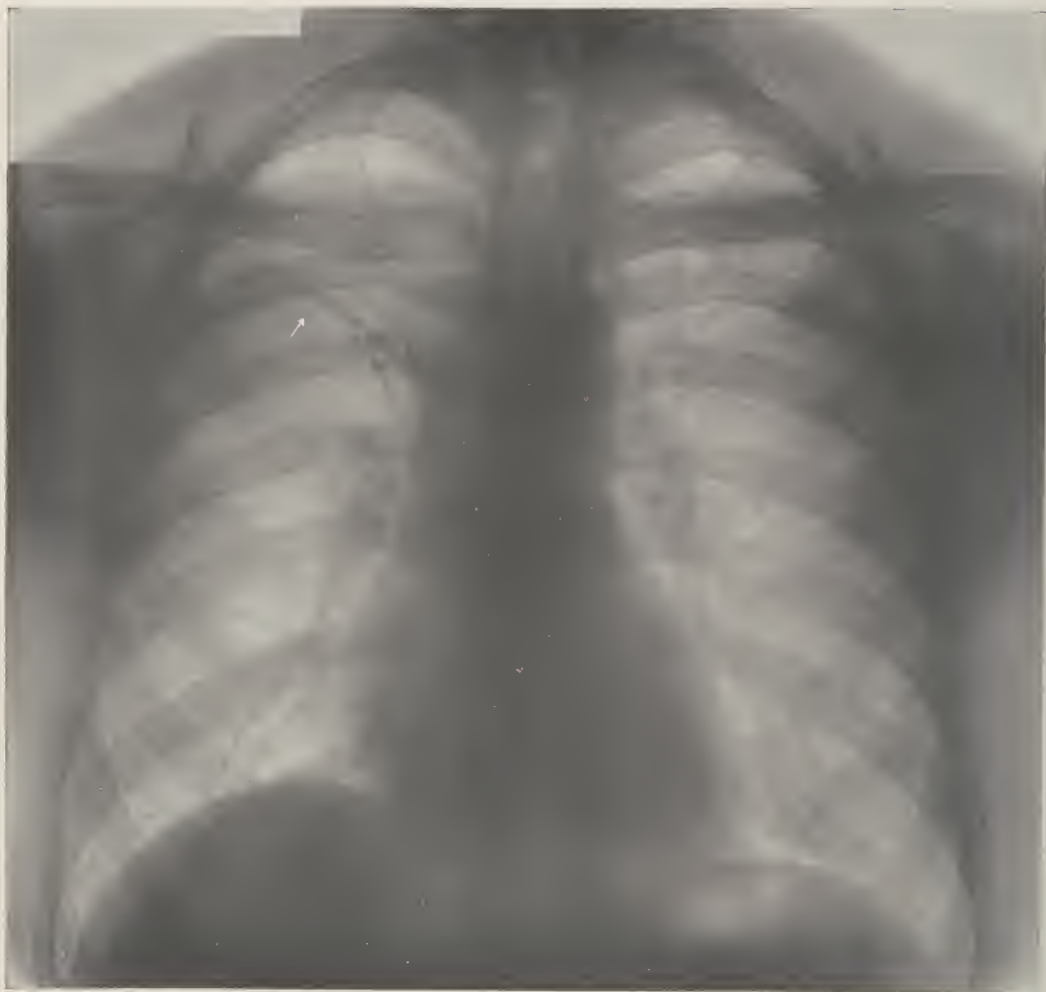


FIGURE 35

Fine fibrosis of healed tuberculosis in right infraclavicular region.



FIGURE 36

Fibrous strand of healed tuberculosis in left upper lobe. (Arrow.)

tissue extends across the upper left chest from the root to the axilla. At times a complicated mosaic may be produced from the interlacing of great numbers of connective tissue strands. (Fig. 37.)

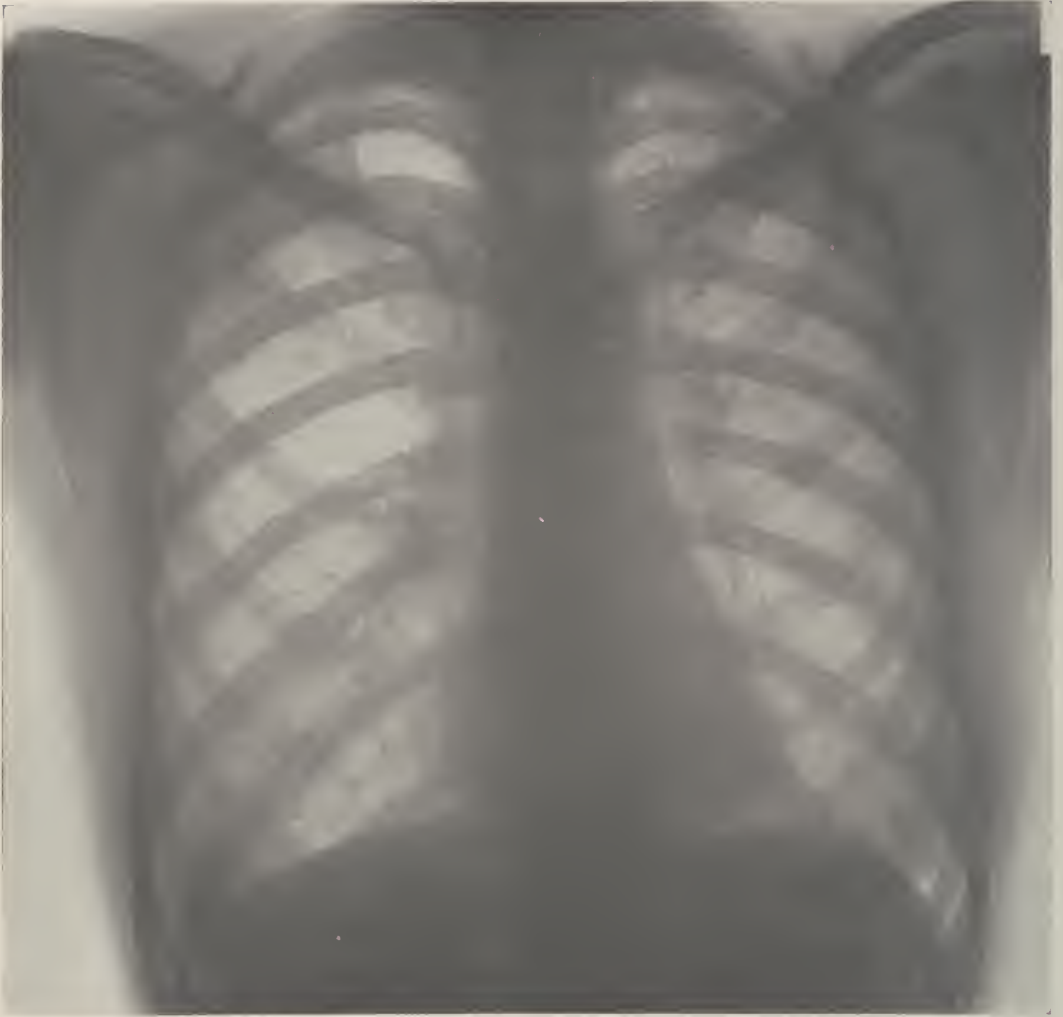


FIGURE 37

Fibrous tissue at left apex producing a unique network of shadows.

In figure 38 we may observe the healing of a more extensive apical process by a coarse fibrosis. The arrangement of the fibrous strands usually conforms in direction to that of the lung markings. In the extreme apex the network is disposed vertically whereas in the lower part of the upper lobe it is oblique or horizontal.



FIGURE 38

Fibrosis of healed tuberculosis at right apex.

The fibrous strands of healing incipient deposits as they have been thus far illustrated, are coarse and arrange themselves in a well defined network, usually limited to a small area in an upper lobe. There is another form of fibrosis in which the lung is over-laid by a fine veil-like network whose strands are so tenuous as to be almost invisible. This may be seen in the lower part of the right upper lobe in figure 39 where the increased density of this part of the lung becomes obvious only by comparison with the opposite, normal lung. It is probable that a network of this type is due both to a fine fibrosis and to a condensation of the lung brought about by obliteration of a number of air vesicles.

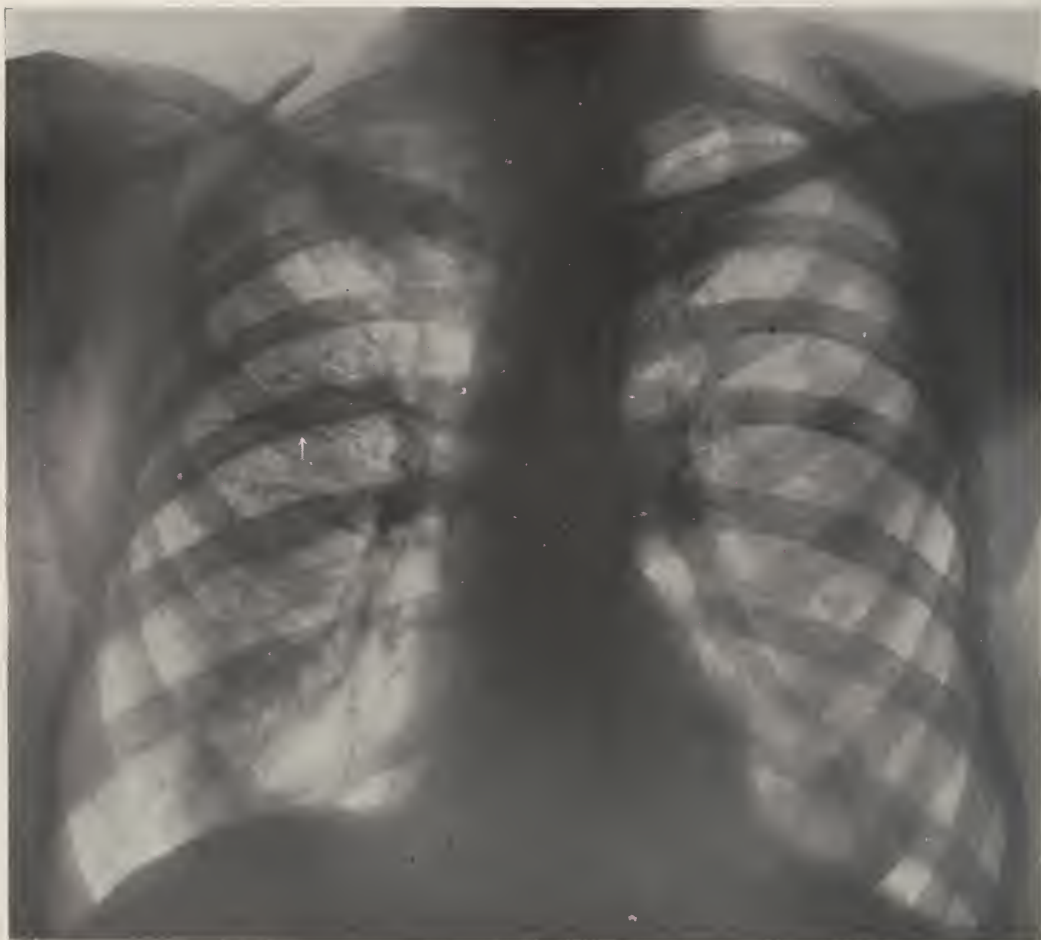


FIGURE 39

Fibrous network in lower part of right upper Thickening of interlobar fissure.

A very unusual manifestation of the healing process is illustrated in figure 40. At the left apex there is a fine network whose vascular nature is so apparent that it requires little effort to believe that the tuberculous process has resulted in an obstruction to the small pulmonary veins or lymphatics whose distention is responsible for the unique Roentgen picture. Here we are leaving the realm of fact for that of conjecture yet we cannot without comment pass by so striking an appearance, even though we can only surmise its meaning.



FIGURE 40

Vascular network at apex probably secondary to healed tuberculosis.

Of course the fibrosing deposits of early tuberculosis may be associated in the same case with other infiltrations in various stages of regression or advance. Thus in figure 41 we may observe in the one plate not only the process of healing characterized by sharply circumscribed shadows of calcified tubercles and fibrosis but also an irruption of recent miliary and sub-miliary tubercles.

Besides fibrosis, calcification of the infiltrations affords us the clearest evidence of cure of the disease. Usually deposition of lime is but an incident in the course of a tuberculous process which is progressing elsewhere in the lung. (Figure 41.) Less often a tuberculous



FIGURE 41

Coincidence of calcified tuberculosis in left lung with recent pneumonic tuberculosis at right apex.

infiltration undergoes calcification in its entirety. The Roentgen shadows are then unmistakable in their denseness and irregularity. (Fig. 41a and Fig. 42.) Bizarre shadows as in figure 43, due to massive lime deposits are very uncommon.



FIGURE 41a

Healed tuberculous bronch-pneumonia, showing disseminated calcified infiltrations.
Scar at right apex.



FIGURE 42
Calcified apical tuberculosis.



FIGURE 43
Calcified tuberculosis.

We must finally discuss certain shadows which are often found at the extreme apices of both healthy and clinically tuberculous individuals, which have the greatest significance for the diagnosis of healed apical lesions. The healing of an apical tuberculosis is often followed by a decrease in the volume of the apex, due either to atelectasis or to fibrosis of the lung and pleura or to both. In the average individual, on a properly exposed plate, aerated lung may be seen as high as the lower border of the second rib posteriorly. (Fig. 44.) With a diminution in the volume of the apex from any cause, the upper limit of the lung is found at a variable distance below its usual situation and the soft tissues at the root of the neck and the subclavian vessels descend and cast a shadow



FIGURE 44

Appearance of normal apices, showing the upper limit of aerated lung.
Dorso-ventral view.

which traverses the apex parallel to, and just below, the second rib.
(Fig. 45.) Such a retraction of the apex is of significance only when



FIGURE 45

Retraction of apices with healed tuberculosis. Fibrous strands dipping down from apical
pleura into lung, on right side.

it is unilateral. When it is found on both sides, its value for diagnosis is limited by its frequent occurrence in individuals who under-ventilate their lungs, either from habit or as a result of nasal obstruction.

A retraction of the apex is often associated with strands of connective tissue which extend downward from its surface into the parenchyma. (Fig. 45.) In figures 46 and 47 are illustrated other forms



FIGURE 46

Retraction and scarring of both apices.



FIGURE 47

Chronic apical tuberculosis. Vertical scar at right apex.

of apical fibrosis. Another cause of apical induration is a thickening of the pleura, which gives rise to dense homogeneous shadows which obscure the apices. Thus in figure 48 the entire left apex is clouded

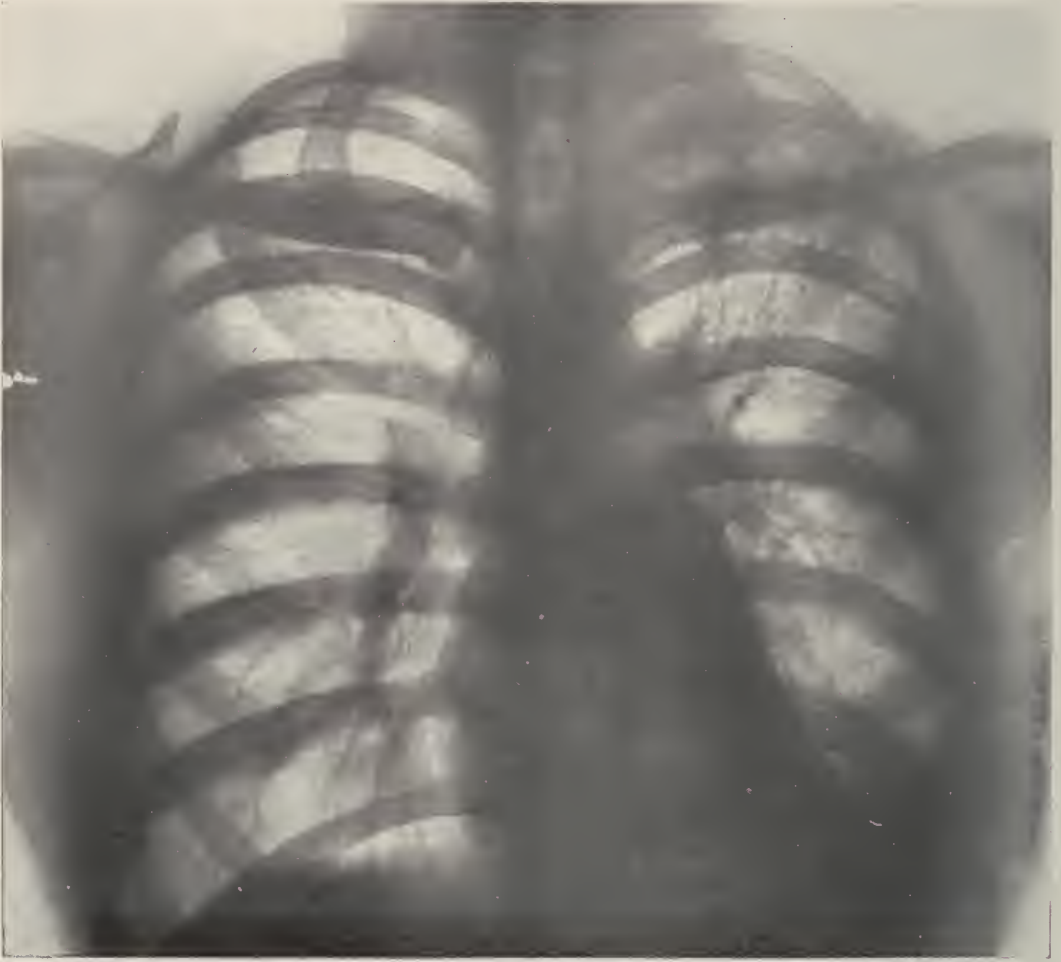


FIGURE 48

Fibrosing process in left upper lobe with thickening of apical pleura and irregular retraction of the apex.

by a thick pleura whose lower margin presents a unique crenated border. Here the apical disease is combined with a fibrosing process below the clavicle. In figure 49 there is a marked induration of the extreme right apex.



FIGURE 49

Fibrosis and retraction of the right apex by old tuberculosis.

The frequent finding of retracted and scarred apices without evidence of tuberculosis elsewhere in the lungs lends some support to the belief that in some of these cases the infection is derived from the cervical lymphatics and runs its course primarily in the apical pleura rather than in the lung. We may thus explain the entire absence of symptoms of antecedent pulmonary disease in many of these cases.

We may profitably dwell for a moment on the clinical significance of retracted or scarred apices especially in respect to the diagnosis of active lesions. These apical changes are so often found in healthy individuals that no clinical significance attaches to them. However, in a number of patients whose apices show the marks of past tuberculous infection, rales are occasionally heard and it becomes a matter of importance to determine whether these rales indicate an active process or not.

Of course, in resolving this question, the clinician is governed not alone by the physical signs but also by the clinical signs of activity. The significance of apical rales taken by themselves may easily be exaggerated. We have thus found in many cases, in which rales were heard at the apices, nothing more than the retraction and induration of an old tuberculous process and it is fair to assume that these rales were not any evidence of activity but were a result of atelectasis or of inflammatory processes other than tuberculosis. We have, on the other hand, found that whenever rales at the apices are due to an active, recent tuberculosis, the infiltrations which we have described as characteristic of early tuberculosis are present in the apices of the lungs.

THE EVOLUTION OF THE TUBERCULOUS PROCESS FROM ITS INCIPIENCY

The diagnosis of diseases in terms of pathologic changes in particular organs is the goal toward which the true clinician bends his efforts. Each advance in the art of diagnosis is accomplished by a widening of our knowledge either of the abnormalities of function or of the structure of diseased tissues. To this rule the Roentgen Ray is no exception and it has measurably enriched our knowledge of the gross changes that occur in the lung when it is infected by the tubercle bacillus.

It is not unnatural for the Roentgenologist who is confronted with the great diversity of shadows seen on the plate in cases of pulmonary tuberculosis, to seek a correlation between them and the morbid changes in the lungs with whose pathology he is already familiar. He will not be content with the mere knowledge that the lungs of a patient he has examined contain infiltrations; he will endeavor to ascertain more precisely the nature of these infiltrations and to bring their multiform shadows into relation with definite pathologic processes.

When we pass from a consideration of incipient tuberculosis to the manifold forms of the disease which result from its gradual or rapid extension, we become aware of an alteration in the appearance of the shadows which is dependent on a change in the inflammatory reaction in the lungs. These reactions, comprising three distinct pathologic types, namely, the proliferative, the exudative and the fibrous, are registered on the plate in a manner which is more or less distinctive.

The proliferative form of tuberculosis we have already encountered in our discussion of incipient disease. Its prototype is the tubercle, whether miliary or conglomerate and it manifests itself on the plate as a small, faint, nodular shadow of varying size, which is often surrounded by a pneumonic zone.

The exudative form is represented by the acute inflammatory reaction of a specific nature which is commonly associated with a rapid dissemination of the disease. Its shadows are dense and homogeneous, often involving large areas of the lung. In distribution it may be lobular or lobar, or coalescence of smaller infiltrations may result in a pseudo-lobar infiltration. It probably accompanies to a slight degree most cases of the proliferative type but it may exist alone as an exudate in the air vesicles with no evidence of tubercle formation. Although in occasional cases it is susceptible of resorption, it is usually the precursor of caseation and cavity formation and is ominous in its import. We shall note its presence wherever there is a widespread dissemination of the tubercle bacillus or its products, either through the circulation or by way of the bronchial tree.

The third type of tuberculous tissue is the fibrous, which represents its indurative or healing form. Evidence of it as fine, interweaving, linear shadows may be met with in even early lesions. It is to be seen to some extent in the plates of all but the most acute forms of tuberculosis, such as the miliary and acute tuberculous pneumonia. In the more chronic cases, fibrous tissue will be associated with evidence of destruction of the lung parenchyma such as caseation and cavity formation. It then appears usually as dense bands of scar tissue or as coarse strands, the results of whose contraction will be noted in a retraction of the lung, displacement of the thoracic viscera and deformity of the bony thorax.

Two important secondary changes in tuberculous infiltrations remain to be described, namely, caseation and calcification. The former especially exerts a marked influence on the types of shadows which are produced; it manifests itself by an increase in their density and in the case of the proliferative lesions, by an added definiteness of outline. The dense, homogeneous shadows of lobar and confluent lobular pneumonia are partly at least, the result of caseation. Calcification of tuberculous infiltrations is always an indication of chronicity and may be looked upon as the best evidence of healing or of a tendency to it. The shadows are circumscribed, very dense and irregular. Lime deposits may present the only vestige on the plate of a previous tuberculous process; more often, however, they are found in conjunction with evidences of more recent disease and we will occasionally encounter foci of calcification within an area of pneumonic tuberculosis.

MODERATELY ADVANCED TUBERCULOSIS

We are now prepared for a consideration of moderately advanced tuberculosis as it is revealed to us on the Roentgen plate. It will be found in practice that few patients come under observation during the early stage of the disease which we have already described. It is probable that in a great many, this period of minimal infiltration is unassociated with any distinctive symptoms or physical signs and that it goes on unnoticed to quiescence and cure. That this is so and that the particular manifestation of the infection which brings the patient to the physician's attention is often not the first invasion of the disease is attested by the frequent discovery, side by side with a recent infiltration, of a healed lesion elsewhere in the lungs. Thus in figure 50, the area of pneumonic tuberculosis in the right upper lobe was directly responsible for the patient's symptoms and physical signs, yet in the opposite apex and infraclavicular region there are unmistakable signs of a calcified tuberculous infiltration which was in process of cure, and which had never given rise to any symptoms.



FIGURE 50

Exudative tuberculosis of right lung with evidences of old, calcified lesions in both upper lobes.

It therefore happens that in many patients who for the first time present symptoms and signs of tuberculosis, we already find so considerable an area of infiltration in an upper lobe, that, from the Roentgen viewpoint at least, it is no longer permissible to regard them as incipient cases. The shadows which characterize this stage of the disease are in general similar to those of the earliest process, except that they are of greater extent and are apt to be denser and more irregular owing to beginning fibrosis or caseation. Figures 51, 52, 53 and 54 illustrate only a few of the many forms which these moderately advanced cases may assume.



FIGURE 51

Pneumonic type of tuberculosis, with signs of moderate fibrosis in upper lobe. Heart and mediastinum drawn to right. Adhesion of mesial part of right diaphragm.



FIGURE 52

Apical tuberculosis; disease is moderately advanced, showing denser shadows of caseation.



FIGURE 53

Acute pneumonic type of tuberculosis; old cavity at left apex.



FIGURE 54
Moderately advanced tuberculosis.

It is of interest to inquire into the manner in which these moderately advanced infiltrations arise in the lung. It is unlikely that they are evolved by a gradual extension of the small deposits of incipient tuberculosis because the latter exhibit a marked constancy in size when observed over extended periods and do not appear to increase in size by peripheral growth. It is more probable that from the beginning the disease is of greater extent, a small initial focus being rapidly converted into a large one by an irruption of tubercle bacilli into the bronchial tree or into the circulation. We are confirmed in this conjecture by the appearance of the Roentgen shadows, which often indicate the operation of a more acute exudative process.

THE ROENTGEN FINDINGS OF ADVANCED TUBERCULOSIS

The transition from tuberculosis of moderate extent to the more advanced forms in which larger areas of the lungs are brought under the influence of the disease, brings us face to face with an embarrassing wealth of material which taxes the power of description. We will find it both necessary and advantageous to depart from the traditional classification of Albrecht and Turban because they cannot be made to harmonize with the Roentgen findings and also because the latter will speak to us in a manner which is more picturesque and instructive.

As we have already pointed out, the spread of tuberculosis in the lungs is accomplished only to a limited degree by a gradual extension peripherally. The major strides of the disease are usually made in a more rapid fashion and their effects may truly be described as catastrophic; a patient who today harbors only a small focus in an apex, may on the next day be seized in the throes of a fatal infection. The means by which this is brought about are the following: (1) hemorrhage, by means of which previously uninvolved areas of the lung become inundated with blood, containing tubercle bacilli; (2) a similar dissemination of tuberculous pus through the bronchi; (3) metastatic invasion by way of the pulmonary blood vessels and lymphatics.

The inflammatory reaction which results from the deposit by these means of large numbers of tubercle bacilli or their toxic products in previously healthy lung tissue, is usually an acute one and is manifested as an acute tuberculous pneumonia, which may be lobular or lobar in distribution. We are dealing here no longer with a pathologic process whose normal tendency, as in the apex, is toward fibrosis but on the contrary with one which is prone to undergo caseation and softening and to form cavities.

When we therefore analyze the Roentgen shadows in cases of advanced tuberculosis, there will always be noted at one or both apices evidence of the original process from which the more recent acute

infiltrations have been derived. The former is often in a condition of partial fibrosis and frequently discloses a smaller or larger cavity surrounded by infiltrations. The shadows of more recent involvement of the lung will then be found in more remote regions in the upper or in the lower lobes, either on one side or on both.

A simple illustration of the evolution of advanced tuberculosis may be seen in figure 55. In the right upper lobe, there is a cavity which has probably existed for some time. We see also in the left upper lobe,



FIGURE 55

Advanced tuberculosis. Disseminated tuberculous broncho-pneumonia secondary to cavity in right upper lobe.

undoubtedly due to a more recent involvement, a disseminated lobular pneumonia, the shadows of which are indistinct in outline and show evidence of fibrosis. Again in figure 56 we may observe the pernicious effect of pulmonary hemorrhage. This patient, a young woman, was for some time affected with an apparently benign apical process of small extent. Shortly before she came under observation, she had a large hemoptysis which was followed by a long period of acute illness. We



FIGURE 56

Acute tuberculous broncho-pneumonia with confluence of the infiltrations at right base. Secondary to hemorrhage from an incipient lesion at apex.

see graphically on the plate the disseminated broncho-pneumonia in the upper lobe and the dense shadow of a confluent pneumonic process, which is probably undergoing caseation, at the base. At a stroke, through a complication of a minor tuberculous process, this patient became acutely ill and emerged from her malady a hopeless invalid.

The acuteness of the infection is in a measure reflected in the type of infiltration on the plate. The shadows are dense and flocculent, hazy in outline and irregular in shape and within some of them we can already see small circular defects, due to beginning softening and cavity formation. These small cavities are commonly not visible in the lower lobes, as they are obscured by the dense overlying infiltration; yet under favorable conditions when there is a preponderance of softening, they may come to light in great numbers and they will then impart a moth-eaten appearance to the lung. (Fig. 78.) The larger cavities which are associated with fibro-caseous tuberculosis result from the breaking down of extensive areas of pneumonia and they may excavate a whole lobe or an entire lung. (Fig. 64a.)

The extent of the pneumonic reaction which immediately follows a hemorrhage or the diffusion of purulent secretions from the bronchi, does not always afford a just estimate of the permanent damage to the lung. After a short interval there will often be noted a decrease in the size and density of the infiltrations which must be attributed to a resorption of a part of the exudate. In fact there are occasional cases, to which we shall refer later, in which the exudate is of such a character, that caseation does not set in but complete resorption occurs. In this form of pneumonia, which is often associated with the overflow of the contents of an apical cavity, very few tubercle bacilli may be found and the pathologic process may be regarded as a reaction to the toxic products of the tubercle bacillus rather than to the micro-organism itself.

The Roentgen plate depicts not only the acute process which is running its course at that particular time but registers also the effects of previous similar invasions of the disease. Tuberculous lobular, or broncho-pneumonia does not invariably go on to softening. The disease may become quiescent or caseation may progress slowly hand in hand with fibrosis so that the clinical picture of fibro-caseous phthisis is evolved. Meanwhile another accession of pneumonia may extend the process elsewhere in the lungs. We may observe this progression in figure 57. The right upper lobe presents the typical picture of a chronic fibro-caseous tuberculosis with a large cavity at the apex. We can dimly see the horizontal strands of fibrous tissue; the marked reduction of this lobe to at least one-half its former size is apparent. We perceive here the evidence of an old pneumonic process in which the elements of healing and destruction are contending with each other. At the



FIGURE 57

Disseminated lobular (broncho-) pneumonia, secondary to chronic fibro-caseous process in right upper lobe. Note shrinkage of right upper lobe as a result of fibrosis.

same time, in contra-distinction to this, there is revealed in the left lung a more recent, extensive lobular pneumonia, which no doubt owes its origin to the older process on the right side.

A study of these advanced forms of the disease which we have illustrated cannot fail to impress the observer with the menace of an unhealed apical lesion and the unfortunate consequences to the patient which result from its activity. It would, however, present our subject in an altogether too gloomy light, were the results of a rapid extension

of the morbid process conceived as leading invariably to a hopeless issue. There is ample proof that in many cases the reaction in the lungs following a dissemination of the disease, may be a relatively mild one in which proliferative processes may dominate the exudative and necrotic. We are dealing here with the delicate balance between the virulence of the tubercle bacillus on the one hand and the local tissue resistance on the other, in regard to which we can only speculate. The fact remains, however, that in numerous cases of even widespread disease, the plates show considerable evidence of absorption of the



FIGURE 58

Bilateral upper lobe tuberculosis showing marked tendency to fibrosis.

broncho-pneumonic areas and of different grades of fibrosis. Figure 58 illustrates a common Roentgen type in which both upper lobes and at times the lower lobes, show marked signs of fibrosis, in spite of the presence of an extensive broncho-pneumonic tuberculosis. Such patients may remain in good health under favorable conditions for many years, with all the clinical signs of a complete arrest of activity. We may therefore in some cases speak of a true chronic lobular or broncho-pneumonia in which the interplay of the conservative and destructive influences in the lung is so evenly adjusted that the progress of the disease is almost imperceptible. An instructive instance may here be cited.



FIGURE 59

Chronic miliary tuberculosis in a diabetic, of at least five years duration. See fig. 60.

A middle aged diabetic woman had suffered for several years with fibrinous bronchitis which was apparently the only manifestation of her pulmonary disease. The Roentgen examination, figure 59, showed an extensive tuberculous process, sub-miliary in type, which involved both lungs from apex to base. The infiltrations were fairly distinct and closely studded and an immediate impression of a rapidly fatal tuberculosis was created; yet, six years later this patient was still alive and in satisfactory health. As a more recent plate illustrates, (fig. 60), the areas of lobular or broncho-pneumonia had to a great extent been absorbed or replaced by fibrous tissue and at the same time a slow disintegration had occurred at the apices where two cavities were formed. Yet this destructive process progressed so insidiously that at no time has she been acutely ill or had other than minor pulmonary symptoms.



FIGURE 60

Same case as fig. 59 six years later, showing disseminated fibrosis and formation of apical cavities.

FIBRO-CASEOUS AND FIBROID TUBERCULOSIS

The ultimate outcome of advanced tuberculosis, as far as the integrity of the lungs is concerned, is determined by the constant activity of two opposed processes, namely, caseation and fibrosis. This conflict is often protracted over many years, during which the opportunity is given us to observe on the Roentgen plate a series of variegated pictures. At one end of the scale we observe the predominantly caseous form of the disease, at the other, the purely fibroid type and between them all gradations of each. In figure 61 is illustrated a typical example of a fibro-caseous process. The disease involves the entire left lung

**FIGURE 61**

Chronic fibro-caseous tuberculosis with cavities. Contraction of left chest and displacement of heart and mediastinum to left.

in a massive consolidation in which there are distinct signs of multiple cavitation. The fibrosis is manifested in the usual way by shrinking of the chest and displacement of the heart and mediastinum to the left. As is usual in these cases, there is also an involvement, though slight, of the other lung. In figure 62 we may observe a greater degree of fibrosis associated with caseation. The remarkable diminution in the size of the right lung at once arrests attention; it is roughly one-half the volume of the left. The trachea and mediastinum are drawn over to the right side by adhesions which have also involved the phrenic nerve. The paralysis of the latter is responsible for the abnormally high diaphragm which further reduces the volume of the chest. Again in figure 64a fibrosis has extensively involved the right lung and has induced a marked derangement of all the thoracic viscera. In this case, however, caseation and softening have kept pace with the fibrosis, with the result that almost the entire right lung is excavated by irregular, trabeculated cavities.



FIGURE 62

Chronic fibro-caseous tuberculosis. Note decrease in volume of right chest and displacement of heart and mediastinum. Right diaphragm in paralytic position.

Of course, fibro-caseous tuberculosis may involve only a restricted area of the lung, such as an upper lobe and it may thus persist for years, presenting the common clinical picture of chronic bronchitis with seasonal exacerbations. Often these patients acquire a secondary emphysema, the physical signs of which may completely mask the underlying tuberculosis and we are then apt to find such a Roentgen picture as figure 63. Here belongs the so-called asthmatic form of



FIGURE 63

Dense fibro-caseous tuberculosis of right upper lobe with cavity. Recent tuberculosis in right lower and left upper lobes.

tuberculosis in which, especially in older individuals, an indurative process at the apices, which is often latent, is probably the cause of both bronchitis and asthma. (Fig. 64.)

Chronic fibro-caseous tuberculosis at the apex is also responsible for the numerous cases of a solitary cavity in this situation which is surrounded by a dense fibrous capsule.



FIGURE 64

Chronic fibroid tuberculosis of both upper lobes.

Although pure or nearly pure fibrosis occurs in localized areas in the various forms of tuberculosis we have described, a disseminated pure fibrosis is one of the rare manifestations of the disease. It is true that an extensive fibrosis may develop in the process of healing of a tuberculous broncho-pneumonia, yet it is nearly always combined with



FIGURE 64a

Chronic fibro-caseous tuberculosis with extensive excavation of right lung. Recent process in left lung. thickening of pleura, displacement of mediastinum and thoracic asymmetry.

the evidences of a more acute process. Therefore, when we speak of fibroid tuberculosis, we do so to indicate a preponderating tendency to the formation of fibrous tissue and in this sense, figure 65 may well illustrate this tendency. The entire lungs are here interwoven with a network of more or less linear shadows through which may be dimly discerned the nodular infiltrations of a less chronic process. It will be noted that in this case there is also a large irregular cavity, which

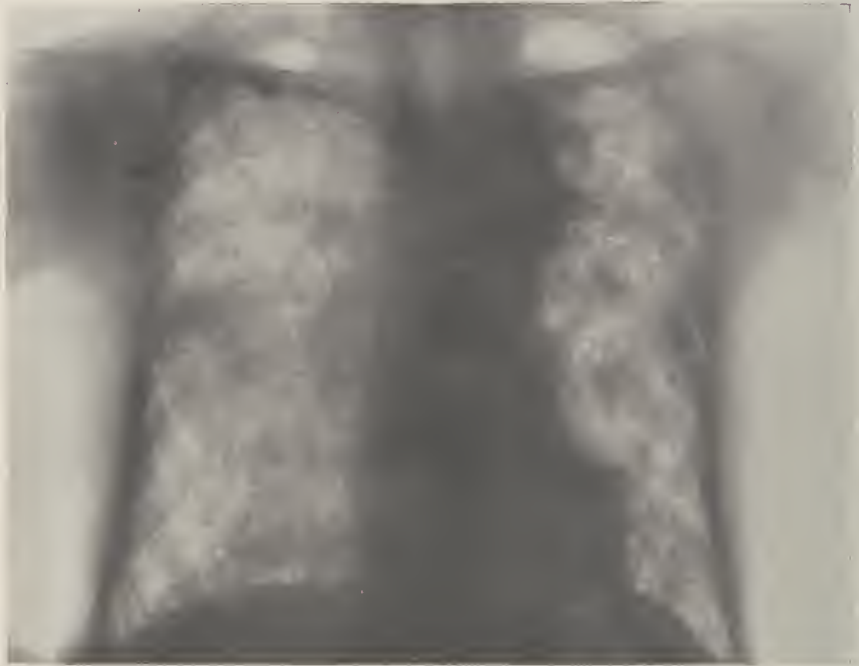


FIGURE 65

Disseminated pure fibrosis. Large cavity in left apex.

occupies the entire left apex and is probably responsible for a constant re-infection of the lung. In figure 66 a wide-spread coarse fibrosis is associated with other evidences of a chronic lesion, consisting of calcification of multiple broncho-pneumonic areas.

Finally, we come to the rare cases of pure disseminated fibrosis of the lung, the diagnosis of which must always remain uncertain. The fine, universal fibrosis of this disease is indistinguishable on the plate-



FIGURE 66

Fibroid tuberculosis in a patient with emphysema and asthma. The punctate shadows represent the very rare calcified tuberculous broncho-pneumonia.

from that which results from the inhalation of particles of mineral matter in some occupational diseases. In both cases the lung is the seat of a chronic, more or less benign inflammatory process. The normal mottling of the lung is replaced by a fine or coarse network which differs from the normal lung in its greater definition, the irregularity and density of its shadows and the presence at an apex of a primitive lesion. The relation of anthracosis to tuberculosis is still unsettled and in the absence of clinical data in a case, the Roentgen plate will provide no reliable points of distinction between these two conditions.

THE ACUTE FORMS OF PULMONARY TUBERCULOSIS

The almost universal tendency of tuberculosis in adults, at least in its early stages, to undergo fibrosis determines from the beginning, its chronic character. Only in its later development, owing to what may be regarded as fortuitous circumstances, does the process at times take on a sudden activity which will convert an indolent infection into an active one.

Tuberculosis may however, assume an acute course from the very beginning which may be marked by an excessive toxemia out of all proportion to the extent of the changes in the lungs. As a rule, the infection in these cases is blood- or lymph-borne, the tubercle bacilli being deposited in the lungs in large numbers from a focus either in the lung or in a remote part of the body.

Acute tuberculosis occurs in two forms, each of which has a distinct pathology and Roentgen appearance. The first we speak of as miliary tuberculosis, which in the one case is a general infection which attacks every organ of the body and in the other is confined to the lung. The pathological process in this group of cases is a proliferative one. The second form comprises the various tuberculous pneumonias, in which the pathological process is essentially an exudative one.

(1) ACUTE GENERAL MILIARY TUBERCULOSIS

There are few diseases of the lungs in which the Roentgen examination exhibits such accuracy and such superiority over other methods of diagnosis as in acute general miliary tuberculosis. At a time when the signs of localization in the lungs have not yet developed and when the disease manifests itself only as a violent intoxication, the plate already reveals an unmistakable and pathognomonic picture. Scattered throughout the lungs from apex to base, filling every lobule, there are myriads of closely studded infiltrations, pin-head in size. (Fig. 67.) In general the infiltrations appear to be more numerous and somewhat larger in the upper than in the lower lobes. The shadows which we see on the plate are not those of individual tubercles but arise from the coalescence of numbers of overlying tubercles. They may be so closely set as to impart a general haziness to the lung fields (Fig. 68), in which individual infiltrations are barely distinguishable. During the course of the disease the shadows increase only little in size; owing to its short duration, caseation has no time to develop and the shadows retain their faintness and lack of definition to the end.



FIGURE 67

Miliary tuberculosis of lungs associated with acute general miliary tuberculosis.



FIGURE 68

Acute miliary tuberculosis. The tubercles are so numerous and closely studded that a general haziness is imparted to the pulmonary fields.

It is characteristic of this disease that, unlike other forms of acute tuberculosis, there is no evidence either in the apices or elsewhere in the lungs, of a previous tuberculous infection. In other words, the tubercle bacillus, transported to the lung by way of the circulation from some focus such as caseous lymph nodes, finds virgin soil for its further development; the disease is therefore not modified or restrained by previous infection.

The clinical advantage of such an accurate diagnostic method is obvious. The physician is now and then confronted by a symptom complex of high fever, sweats and marked toxemia, the cause of which

may for a long time not be apparent. His diagnostic skill is tested to the utmost and the resources of the laboratory may be exhausted in vain to differentiate between such possible causes of the clinical picture as malaria, a general bacterial infection, latent Hodgkin's disease and general miliary tuberculosis. It is possible at once from the typical Roentgen appearance to establish the diagnosis of miliary tuberculosis or by a negative finding to exclude it and thus to narrow the problem.

(2) MILIARY TUBERCULOSIS OF THE LUNGS

In contra-distinction to the disease which we have just discussed we have to consider a miliary infiltration of the lung in which the infection is a purely pulmonary one. Unlike the other, it is not necessarily fatal. Although it is commonly marked in its beginning by the symptoms of a severe infection, it may after weeks or months become quiescent and undergo the secondary changes which are found in sub-acute and chronic tuberculosis. At this point it may be well to clarify our minds in regard to the usage and meaning of the term "miliary" tuberculosis in order that our Roentgen interpretations may, as far as is possible, conform to a pathological entity. Strictly speaking, a miliary tubercle is an agglomeration of epitheloid cells, which is just visible to the naked eye and has a definite microscopic picture. A group of several such tubercles adjacent to each other and coalescent is also often described as a single tubercle. In cases of longer duration, there is often an associated exudative process in the alveoli immediately about them. The symptoms of miliary tuberculosis are due to a widespread dissemination of these units of the disease throughout the lung and the resulting absorption of toxins from them. They may arise in various ways. Probably the most common mechanism is the rupture or growth into a pulmonary vessel or lymphatic of a caseous focus, by which means numbers of tubercle bacilli are deposited in the alveolar walls. The resulting tubercles are not intra-bronchial or alveolar, they are rather interstitial. On the other hand, conceivably, miliary tuberculosis may have a bronchial origin, due to the transport of tuberculous secretions along the bronchial tree. In the exact interpretation of our plates we are therefore confronted with the necessity of distinguishing between so-called miliary broncho-pneumonia and hematogenous or lymphogenous miliary tuberculosis. We may well question the wisdom of drawing our distinctions too fine and of presuming to read too much into our plates. A miliary tubercle or a group of them will cast the same shadow on the plate, no matter what its anatomic situation. For this reason we shall err less in our interpretations if we are guided by the clinical course of the case and by the physical signs. We shall thus expect to find more physical signs of bronchitis in the bronchial form of the disease, whereas the situation of the tubercles in the hematogenous variety will explain the frequent absence of physical signs, even when the disease is extensive.

In figure 69 we see a partial involvement of the lung in a miliary tuberculosis, which is apparently secondary to an older lesion at the right apex. The denser shadows surrounding several small cavities in



FIGURE 69

Miliary tuberculosis of left upper lobe secondary to an old tuberculosis at right apex.

the right upper lobe are in marked contrast to the numberless miliary infiltrations in the rest of the lungs, which represent a recent acute miliary tuberculosis.

A more universal process is illustrated in figures 70 and 71. A significant feature may be observed in many of the tubercles. They are larger and denser, indicating a greater age than is possible in an acute general miliary tuberculosis and in places there are signs of an inflam-



FIGURE 70

Miliary tuberculosis of the broncho-pneumonic or lobular type. Note older lesions at apices with small cavity behind left clavicle.



FIGURE 71

Miliary tuberculosis of broncho-pneumonic type. The individual shadows are larger owing to exudation about the tubercles. At left base the infiltrations are confluent.

matory reaction in the alveoli about them; they may perhaps be better described as submiliary in size. The great similarity exhibited by these two Roentgen plates might lead us to anticipate a correspondence in their clinical pictures. Lest we indulge in such an inference, be it set down that these patients presented the greatest clinical diversity. In the one, figure 71, death ensued in a short time from an overwhelming

toxemia. In the other, figure 70, the patient recovered at least for the time being. He was up and about, and only mildly ill at the time of the examination. Nor in respect to the character of the physical signs does the Roentgen plate offer any basis for judgment. It is remarkable that such extensive infiltrations may be productive of few or no physical signs. The vicarious emphysema of the adjacent uninvolved lung may effectually mask all dulness and may even give rise to a tympanitic note. Rales are often absent during the stage of tubercle formation and may only appear with the development of an exudative inflammation about them.



FIGURE 72

Beginning of an acute tuberculosis which completely involved the lungs in three weeks. Process began in right upper lobe where a moderate number of submiliary infiltrations are seen. See figure 73.

The opportunity is rarely afforded to observe on the Roentgen plate, the very beginning of such acute disseminated processes because at their onset there may be no localizing signs or symptoms in the lungs to direct our attention to them. For this reason, the following case, illustrating graphically the gradual evolution of an undoubtedly hematogenous tuberculosis is of absorbing interest.

The onset of the illness in this boy of 14 years who was previously in good health, had all the signs of an acute infection, including very high fever and prostration. There were neither cough, dyspnoea nor other symptoms to indicate a disease of the lungs. During the



FIGURE 73

Same case as figure 72 one week later, showing increasing number of infiltrations. See figure 74.

period of observation which extended over six weeks, there were remittent fever and progressive anemia; no physical signs except latterly a few small, scattered rales. The plates taken at weekly intervals, a few of which we reproduce, clearly portray the life story of a submiliary tuberculosis.

It will be noted that in figure 72, the first plate made at the onset of the infection, only a few small shadows are visible in the right infra-clavicular region. In the next plate, figure 73, more and more infiltrations make their appearance, becoming also more distinct, until finally in figure 74 we have the complete picture of a disseminated tuberculosis. At this point the temperature subsided and at least temporarily the disease became quiescent.



FIGURE 74

Same case as figure 73 one week later, showing lungs closely studded with innumerable submiliary tubercles.

While the Roentgen examination can thus play an important role in the clarification of the pulmonary features of acute tuberculous infection, it is worth bearing in mind that it may be equally valuable in a negative sense. There are rare cases of generalized glandular tuberculosis in which, associated with an enlargement of single lymph nodes or groups of them, a high temperature and other symptoms of toxemia may continue for long periods. In these cases there is always present the disquieting thought that there may be a concomitant miliary tuberculosis of the lungs. It is apparent that the assurance that the lungs are uninvolved, which only the Roentgen plate can give, will encourage the physician in a more favorable prognosis.

(3) ACUTE PNEUMONIC TUBERCULOSIS

We have already referred to the various lobular and pseudo-lobar exudations which are secondary to existing tuberculous lesions. In most cases, these pneumonias are incidents in the course of a more or less chronic process in the upper lobes and their connection with the more primitive infection is usually obvious both to the clinician and to the Roentgenologist.

Of a different nature, at least clinically, are those cases of tuberculosis which, beginning acutely, in previously healthy individuals, run their course as a pneumonia which is often rapidly fatal. Although it is true that frequently a history of previous pulmonary disease may be obtained, the evidence of which is unmistakable on the plate, this is often so remote that it will not appear to have any relation to the patient's immediate illness.

As is well known, tuberculous pneumonia during its early stage may closely simulate a lobar pneumonia and only the persistence of the symptoms and signs for weeks or even months or the discovery of tubercle bacilli in the sputum will supply a clue to the correct diagnosis. Neither may the Roentgen examination disclose anything distinctive unless there is a rapid softening of the caseous exudate with cavitation. Even then, a cavity may be completely obscured by the overlying consolidation. The Roentgen appearance of a tuberculous lobar pneumonia of the right upper lobe is illustrated in figure 75, in which cavity formation is already indicated. The history was of three weeks' duration, with symptoms indistinguishable from those of lobar pneumonia. Of significance latterly was the expectoration of the green viscid sputum, often regarded as characteristic of gelatinous pneumonia.

The acute tuberculous pneumonias are more often lobular in type consisting of irregular, widely disseminated areas of exudation, whose



FIGURE 75

Caseous pneumonia showing an irregular area of softening at right apex.

coalescence frequently results in a pseudo-lobar appearance. (Fig. 76.) Physical signs, especially moist and metallic rales are found in great profusion with early signs of cavitation. In respect to the latter the physical examiner enjoys an advantage over the Roentgenologist, on whose plates the cavities may long remain unseen, hidden within the dense shadow of the pneumonic exudate. When they become visible, they are usually irregular, appear punched out of the infiltration and grow rapidly in size.

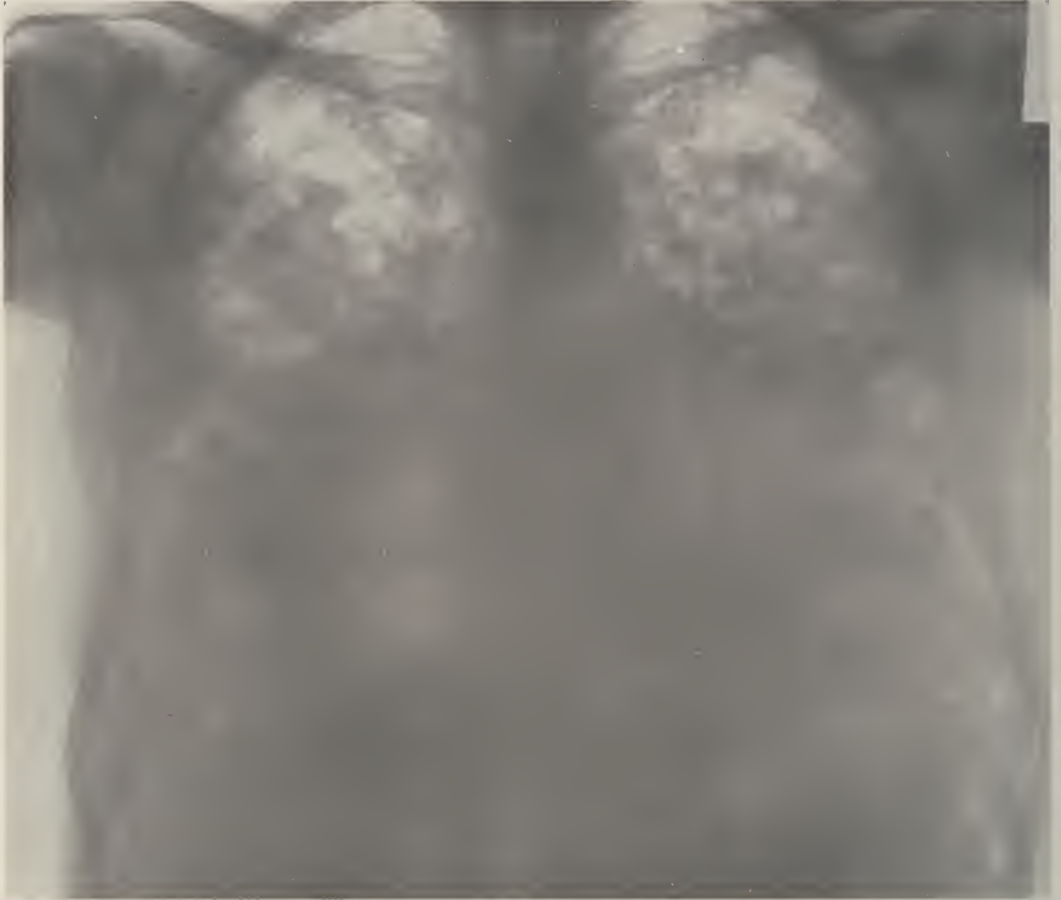


FIGURE 76

Confluent lobular tuberculous pneumonia. Clinical picture of gelatinous pneumonia.

The clinical course of tuberculous pneumonias is usually progressively downward and is characterized by a rapid destruction of the lung parenchyma. We may thus observe on the plate the ravages of the disease, unstayed by any effort at fibrosis. Whole lobes may be excavated en masse leaving only a shell of tissue about the cavities, figure 77,



FIGURE 77

Acute tuberculous pneumonia with rapid and extensive excavation of left upper lobe. Shows trabeculation.

or again the lung may be so riddled with smaller cavities that it appears moth-eaten. (Fig. 78.)

There are however other cases which begin equally acutely, yet, after months of illness the infection takes a subacute course until finally the clinical picture of chronic pulmonary tuberculosis is evolved. The Roentgen shadows are here also extensive and there may be considerable softening, yet there soon appear signs of fibrosis which is more or less effectual in checking the spread of the disease.



FIGURE 78

Disseminated lobular tuberculosis with small areas of softening, producing honey-comb appearance.

Although tuberculous pneumonia, as a rule is ultimately, if not immediately fatal, its clinical course offers many surprises to the physician. At times the course of the infection is stayed and extensive consolidations resorb almost as rapidly as they arose, before the onset of caseation. (Fig. 100.) More remarkably, in cases in which caseation has begun, in which both on the plate and to the ear, signs of extensive cavitation are evident, the disease may come to a halt and the patient who has wavered at the brink of death, rapidly and completely recovers, while subsequent plates show the disappearance of both infiltrations and cavities. (Figs. 103 and 104.)

TUBERCULOUS CAVITIES

The important role which cavities play in the progress of the tuberculous infection, warrants a more minute description of their Roentgen features. There is perhaps no phase of this disease upon which the Roentgen examination has shed more light. It has been of special value in the diagnosis of the smaller cavities, the occurrence of which in even early tuberculosis has been shown to be much more frequent than was supposed. It has taught that the classical amphoric signs of cavities are distinctive only of the larger ones, whereas the great majority, which are small, do not betray their presence by abnormal physical signs.

The favorite site for the development of cavities, namely, the apices and upper lobes, facilitates their recognition on the plate because they are here superficially situated. It is important to realize that cavities in this region may be found at a relatively early stage of the disease and that the clinical classification of a case as incipient may have to be revised and the prognosis correspondingly changed after the discovery of a cavity by the Roentgen Ray.

Thus in figure 79, although as far as the extent of the process in the upper lobe is concerned, this case may be regarded as incipient, there is already a very small cavity at the left apex whose influence on the future course of the infection must be reckoned with.

The appearance of tuberculous cavities depends both on their age and on the nature of the pathologic tissue in which they are formed. In general we may recognize three forms, with minor modifications which are due to the transition of one form into another.

The first and perhaps most common type is the annular cavity, which is found especially in early tuberculosis and represents probably an early stage in its evolution. It appears to result from a slow breaking down of the more chronic proliferative type of infiltration, in which caseation is not extensive and is gradual in its development. It there-

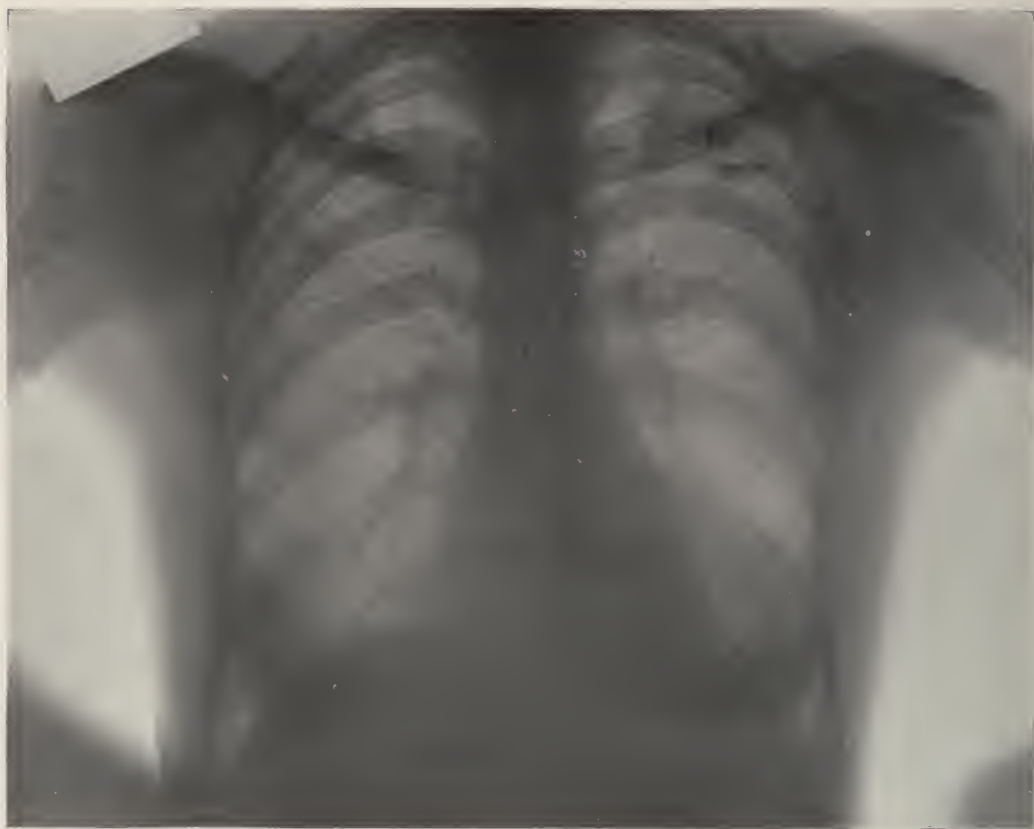


FIGURE 79

Cavity in left upper lobe, showing fluid level, in the course of tuberculous process of small extent.

fore lies in lung which is not densely infiltrated and may often only be recognized by a thin line of demarcation from the surrounding lung. (Fig. 80.) The cavity in the left apex in figure 60 was thus slowly formed during the course of a chronic submiliary tuberculosis which was under observation for a number of years and in which the destruction of the lung parenchyma was a very slow one.

It is probable that in some cases at least, these annular shadows represent a zone of demarcation about an infiltration which will eventually result in a cavity. Only thus can we explain the occasional absence of air within them and the persistence of lung markings which traverse them.

These cavities are practically always situated near, but not in, the apex of the lung. Their favorite site is in the infraclavicular region near the axilla. In fact it may be stated as a general rule that cavities



FIGURE 80

Annular type of cavity at left apex. At right apex, annular pleural shadow.

of moderate size usually do not occupy the extreme apex. The smallest annular cavities may be only a fraction of an inch in diameter; the largest may attain a size of two to three inches. They usually occur singly. This form of cavity is commonly not discovered by the physical examination for reasons which are obvious on the plate. As it is not surrounded by consolidated lung and, as in many cases, it does not communicate with a bronchus, the characteristic physical signs of a cavity are either not produced in it or are not transmitted to the surface of the chest.

The second type of cavity lies in densely infiltrated lung, in which it probably arises from a rapid breaking down of caseous tissue. It has a punched-out appearance, is air-containing and often shows a

fluid level. (Figs. 55 and 103.) It is surrounded by a dense well defined wall which is composed both of fibrous tissue and the adjacent consolidated lung. The demarcation of these cavities is rendered more striking by the introduction of a small amount of air into the pleural cavity. (Fig. 81.)



FIGURE 81

Two cavities in right upper lobe whose walls are accentuated by pneumothorax.

Like the cavities of the first variety these are also most frequently seen in the upper lobes but they may be found in any situation where there is a rapid softening of the lung. Their shape will be determined by the character of their walls, which in turn will depend on the degree to which a fibrous capsule is formed about them. Thus the rapid necrosis which occurs in acute caseous pneumonia may give rise to an irregular excavation (fig. 77), with no evidence of a limiting membrane. On the other hand, when time is afforded for the formation of a capsule they are usually circular in outline.

It is this form of cavity whose progressive invasion of the adjacent diseased lung is responsible for the cases of extensive excavation of a lobe or a whole lung, which illustrate most exquisitely the destructive tendencies of this disease. Under favorable conditions it is here possible to make out trabeculations in the walls of the cavities. (Fig. 77.) At times they are multilocular and they may assume bizarre shapes. (Fig. 90.)

The third type or fibrous-walled cavity represents the terminal stage of either type described, in which conservative influences limit its extension and attempt to encapsulate it. The indurative tendency of apical disease here manifests itself in its most pronounced form and as a result we commonly find cavities of moderate size at an apex surrounded by a zone of connective tissue of varying depth and density. (Figs. 63 and 82.) They are frequently adherent to the chest wall by a much thick-



FIGURE 82

Fibrous-walled chronic cavity at right apex.

ened pleura, which often interposes an insurmountable obstacle to their collapse by artificial pneumothorax. (Fig. 83.) The great density of their capsule is well illustrated in figure 84. Clinically, these cases are

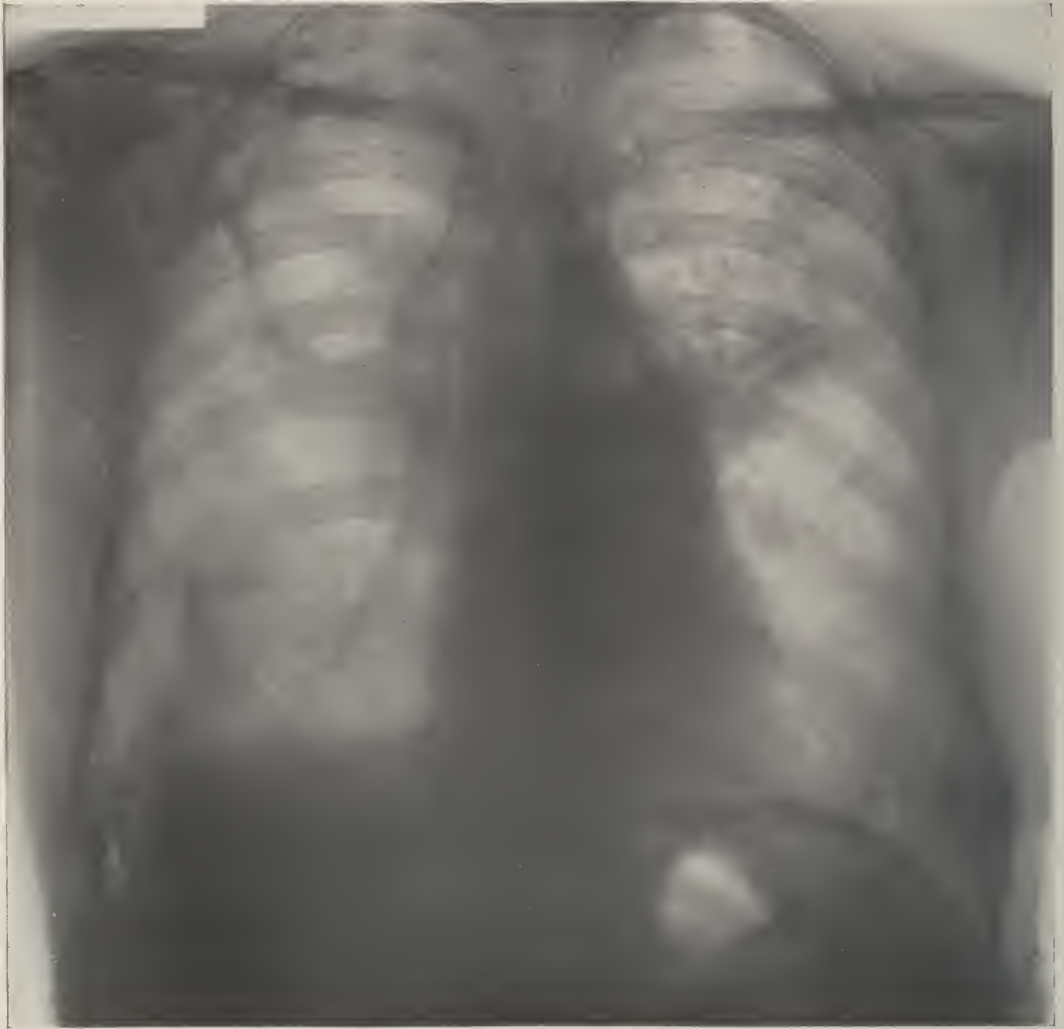


FIGURE 83

Large oval cavity in right upper lobe, adherent to chest wall; pneumothorax.

often characterized by remarkable chronicity. They are consistent with fair health and when, as is often the case, the cavities are completely lined by smooth connective tissue, the expectoration may be negligible and may contain no tubercle bacilli.

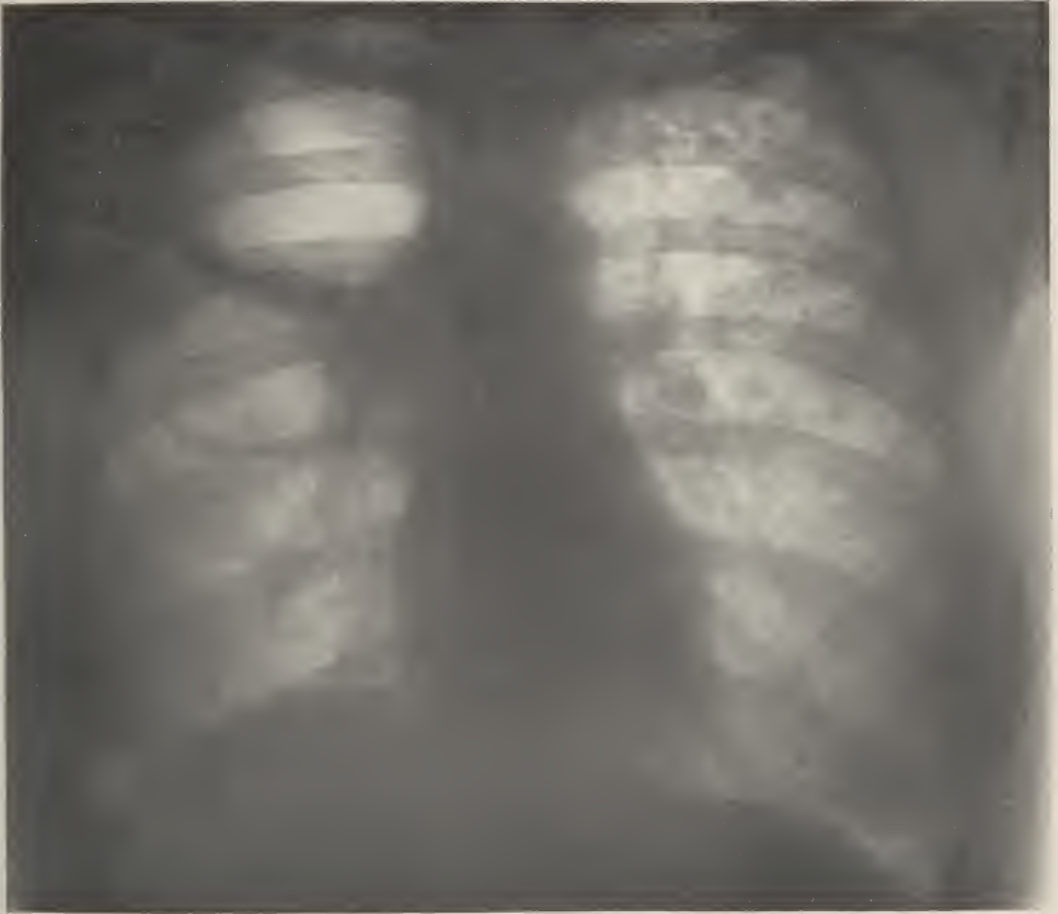


FIGURE 34

Complete excavation of right apex; smaller cavity below.

Although in the upper lobes the Roentgen Ray enjoys distinct advantages over the clinical examination in the diagnosis of cavities, this condition is often reversed in the lower lobes. In the latter location, their greater distance from the surface of the chest and the interposition of infiltrated lung will obscure any but very large cavities. It therefore often happens that the multiple small cavities which so frequently result from the rapid breaking down of a caseous lobular pneumonia are not seen on the plate. In other cases, numerous small circular defects, no larger than a pea may appear throughout the consolidated area, due to multiple areas of softening. Under special circumstances, when such an excavation occurs in patients who survive

this disease for a time, the surrounding infiltration may be absorbed, leaving behind innumerable thin-walled cavities. Thus in figure 78 we have all the evidences of a wide-spread tuberculosis in which necrosis and absorption of the exudate have been so universal and active that the infiltration has almost everywhere given place to cavity formation.



FIGURE 85

Tuberculous cavity in left upper lobe, full of fluid, simulating a consolidation of the lung. See figure 86.

It is noticeable that the contents of tuberculous cavities are not as often represented on the plate by a fluid level as is the case in lung abscess. In the annular type of cavity, a fluid level is rarely seen. It is more common in the punched-out air-containing variety. Even in the very large vomicae which excavate a whole apex it is surprising how

rarely a fluid level is seen on the plate. Under these circumstances we must assume a very complete drainage which prevents the accumulation of secretion. We may occasionally observe the opposite condition in which the cavity, owing to deficient drainage becomes full of fluid, to the exclusion of its air content. A fluid level will then not be seen and the interpretation of the unbroken shadow may encounter insuperable difficulties. The following case illustrates the problem in diagnosis which may be presented under these unusual circumstances:

A middle aged man who for many years had a mild cough without acute illness, began gradually to lose weight and to have a slight daily fever. He presented a cachetic appearance which aroused a strong suspicion of cancer. This seemed to be corroborated by the physical signs in the left upper lobe, which were those of a massive consolidation. The plate, (fig. 85), made at this time revealed a dense homogeneous shadow in the left upper lobe, which plausibly enough was regarded as a carcinoma. During the next few weeks however, the expectoration became more profuse, the temperature subsided and with the evacuation of its contents, the physical signs of a large cavity made their appearance. A second plate now showed a large cavity, at the bottom of which there remained a small amount of fluid. (Fig. 86.)

A word may be said in regard to the interpretation of certain shadows in the lungs, which by a fortuitous arrangement, may simulate cavities. It is not uncommon, especially at the roots of the lungs, for the shadows of blood vessels and indurated lymph nodes to be so disposed as to resemble a cavity wall. It need only be stated that tuberculous cavities at the hilum of the lung are very rare and that a careful scrutiny of these shadows will disclose their true nature. A confusion between the cross section of a bronchus and a cavity is possible only to the inexperienced. Of greater importance than these sources of error, are the annular shadows of pleural adhesions which may be circular in outline and may closely simulate a cavity. (Fig. 80.) The distinction cannot always be made and resort to stereoscopic examination may be necessary in order to determine their true nature.

UNUSUAL TYPES OF TUBERCULOSIS

Most cases of pulmonary tuberculosis as they are revealed to us on the plate, conform to a few recurring types which we have thus far illustrated. With these the Roentgenologist soon becomes familiar and he learns to recognize them at a glance. We shall now consider some atypical forms of tuberculosis whose shadows differ in their location and appearance from the usual and in which, for this reason, the diagnosis cannot so readily be made. The Roentgen examination in these cases is of especial importance because the location of the disease or the nature of the pathological changes themselves may be such as to enhance the difficulties of the physical examination.



FIGURE 86

Same case as figure 85 showing partial emptying of cavity and horizontal level of residual fluid.

HILUM TUBERCULOSIS

By hilum tuberculosis we understand a form of the disease in which the infiltrations are situated at the root of the lung from which they extend outward into the parenchyma for a variable distance, usually however, not to the periphery. It represents therefore a centrally situ-

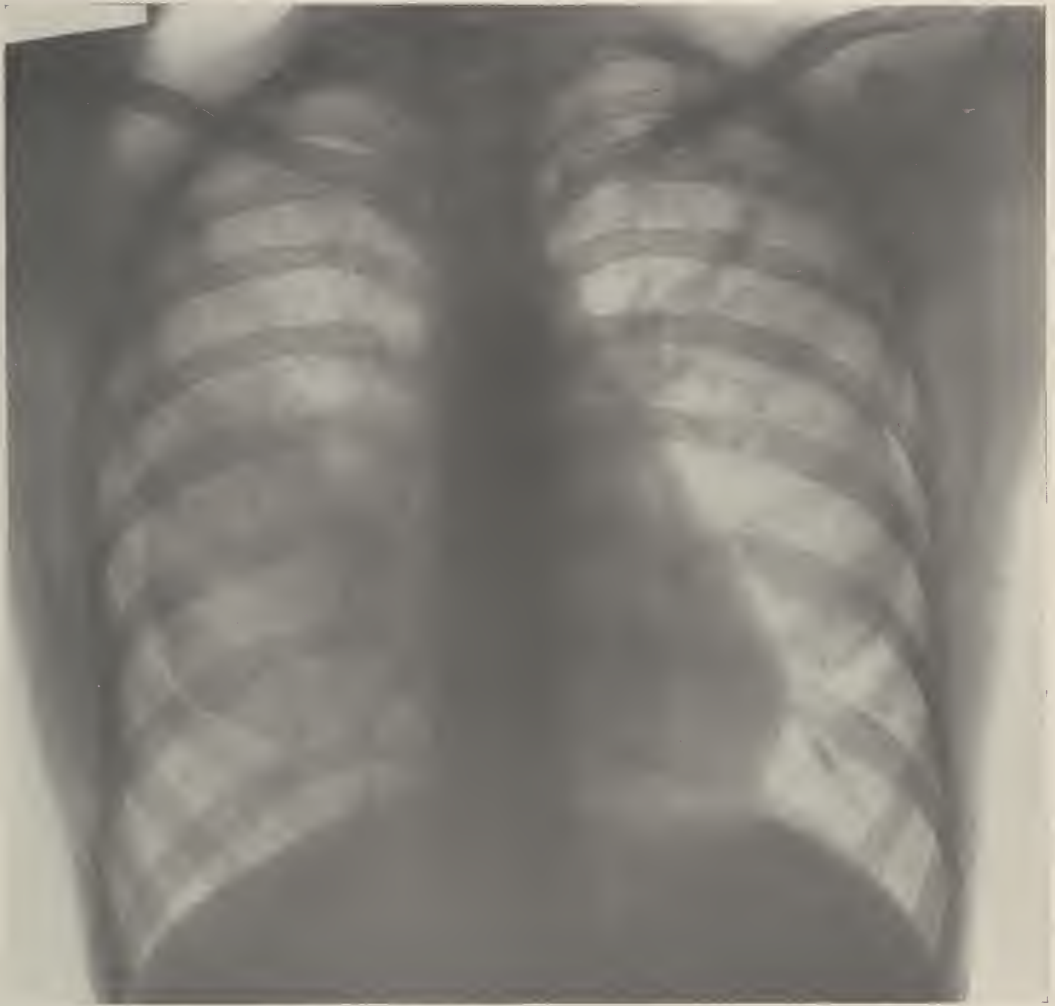


FIGURE 38

Hilum tuberculosis on right side and left upper lobe process.

ated process, the physical signs of which are often obscured by the overlying healthy lung. It should be noted that the term "hilum" is used in a topographical sense and conveys no suggestion as to the pathological process which is present.

It is scarcely necessary to point out that such a localization of the disease is very unusual, so much so, that in adults we have encountered scarcely a dozen cases. What their pathogenesis may be, why the tubercle bacilli are not, as is the rule, deposited in the apical region, we do not know. We may assume as the most likely cause a direct involvement of the pulmonary tissue about tuberculous bronchial lymph nodes. The resulting inflammation may be in the nature of an exudative reaction which is susceptible of resorption, a form of hilum tuberculosis which is more apt to occur in children; or the infiltration may be of the proliferative type and may go on to caseation and cavity formation.



FIGURE 89

Hilum tuberculosis with cavity showing fluid level.

The infiltration more often involves the root of the upper lobe on either side and is seen in characteristic form in figure 88 at the hilum of the right lung. It may be associated as in this case with an apical tuberculosis, a combination which removes all doubt as to the true nature of the condition.

The breaking down of the infiltration at the hilum may result, as elsewhere, in the formation of cavities which consequently will have an unusual location. Thus in figure 89 a small cavity partly full of



FIGURE 90

Hilum tuberculosis on right side with unique clover-leaf cavity.

fluid occupies the summit of the infiltration. In this case, the course of the disease was very rapid, dating back only two and one-half months, during which time the patient was acutely ill and had frequent hemoptyses.



FIGURE 91

Left hilum tuberculosis with unique crescentic cavity.

Figures 90 and 91, illustrate bizarre forms of cavities situated within the hilum infiltration. In figure 92 the infiltration has been almost completely excavated by a cavity.



FIGURE 92

Fibrous-walled cavity. Hilum tuberculosis.

Cases of hilum tuberculosis are remarkable for the paucity of physical signs which may be associated with even an extensive process. The deep situation of the disease and the interposition of healthy lung between it and the chest wall are unfavorable for the propagation of rales or the demonstration of dulness. It will be seen therefore that for the disclosure of this unusual type of tuberculosis, the Roentgen examination is indispensable. Yet it must also be evident that in many cases the tuberculous nature of a root infiltration will not be susceptible of proof by the Roentgen examination alone. In view of the rarity of hilum tuberculosis, other diseases which may involve the roots of the lungs, such as abscess, tumor, or even lung syphilis, must take precedence in the diagnosis. Occasionally the presence of typical tuberculous

infiltrations elsewhere in the lungs or a tuberculous cavity will afford confirmation of its tuberculous nature; otherwise the diagnosis must finally rest on a firm basis of clinical symptoms and signs. We would especially emphasize the desirability of imposing a greater restriction on the use of the term "Hilum Tuberculosis" than has been customary in the past. It has too frequently been applied to the chronic changes, whether tuberculous or indurative, in the bronchial lymph nodes and to an increase in the connective tissue about them, which are almost regularly found in adults. The interpretation of these root shadows as a true hilum tuberculosis has been responsible for the promiscuous diagnosis of tuberculous disease in healthy individuals and lies at the bottom of much of the distrust with which the clinician in the past has viewed the Roentgen findings.

ISOLATED LOWER LOBE TUBERCULOSIS

Although infiltration of a lower lobe is a frequent happening in advanced tuberculosis where it is commonly secondary to an apical process, a primary involvement of the lower lobes belongs to the rarities. For this reason the Roentgenologist will wisely hesitate to predicate the tuberculous nature of an isolated lower lobe process, unless supported by a positive sputum finding.

The character of the infiltration differs little, if at all, from that of non-tuberculous diseases, such as chronic pneumonias or bronchiectases; consequently it is necessary to scrutinize carefully the apices for evidences of tuberculosis, into relation with which the basilar process can be brought. In basal tuberculosis, cavities are usually not visible on the plate, even in the more acute caseous forms of the disease. In deciding whether an isolated basal process is tuberculous, it is important to seek in the clinical history of the case those associated diseases which are known to predispose to a basal process. The most important of these is probably diabetes mellitus which, in the few cases observed by us was its most common precursor.

In figure 93 is illustrated a tuberculous process in a young diabetic which involves the summit and base of the left lower lobe, the intervening tissue being unaffected. The patient's sugar tolerance was very slight; the tuberculous process exhibited physical signs of great activity, all of which were over the left lobe posteriorly.

Other causes which may determine a basal tuberculosis such as kypho-scoliosis and deformities of the chest following trauma, will usually be evident on the plate.

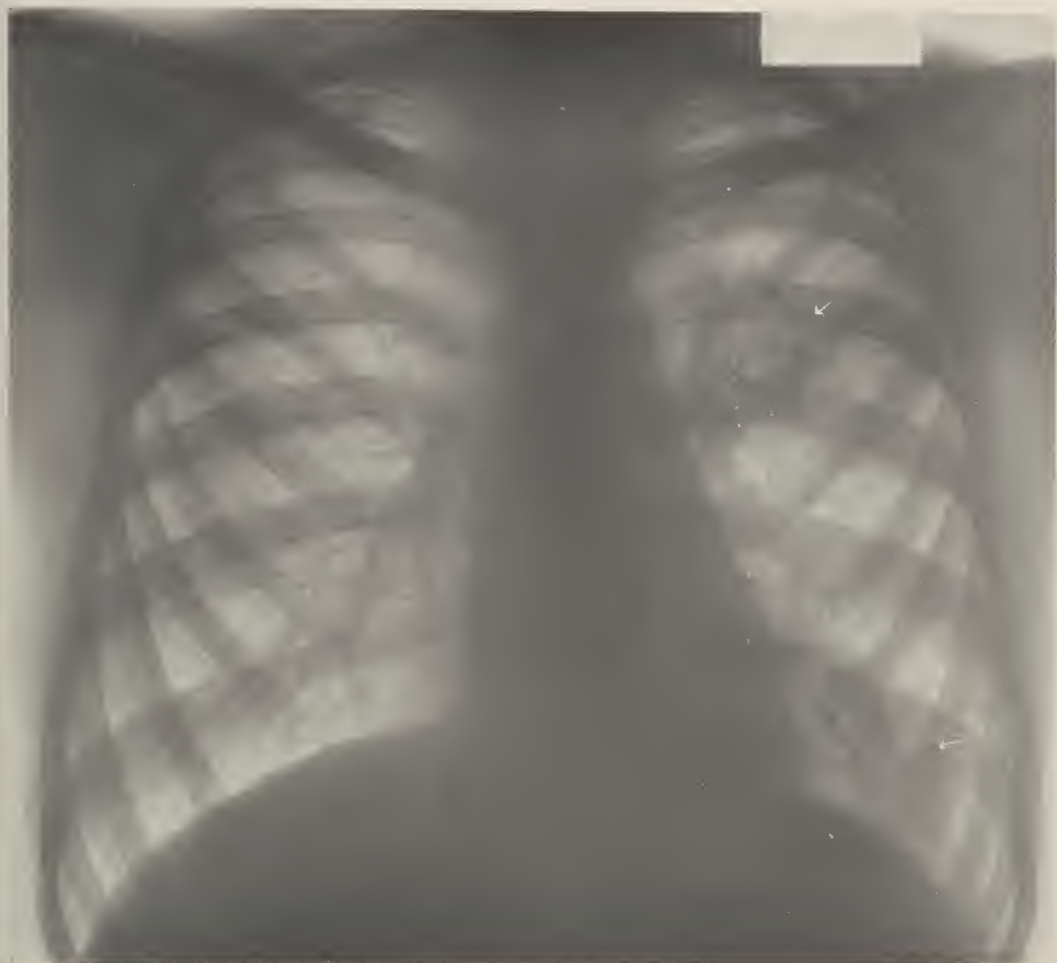


FIGURE 93
Tuberculosis of left lower lobe in a diabetic.

SOLITARY CAVITIES WITHOUT INFILTRATION

The cavities which are found in the upper lobes of cases of chronic pulmonary tuberculosis are practically always surrounded by infiltrated lung. The discovery of a cavity which lies in apparently healthy lung is calculated therefore to arrest attention and it will be proper to inquire into the clinical history for corroborative evidence of tuberculosis, before such a rare manifestation of the disease is presumed to exist.

Yet rare as they are, such cases undoubtedly occur and one must assume that the infiltration which had previously been present was completely absorbed, only the cavity remaining.

Thus in figure 94 may be seen the outlines of a cavity situated to the left of, and behind the heart, which contains a small amount of fluid. Similarly in figure 95 a circular cavity occupies the lower portion



FIGURE 94

Annular cavity near left border of heart lying in normal lung. Patient later developed extensive tuberculosis.

of the left upper lobe. Here the walls of the cavity are not completely developed and it required an examination in the tilted position shown

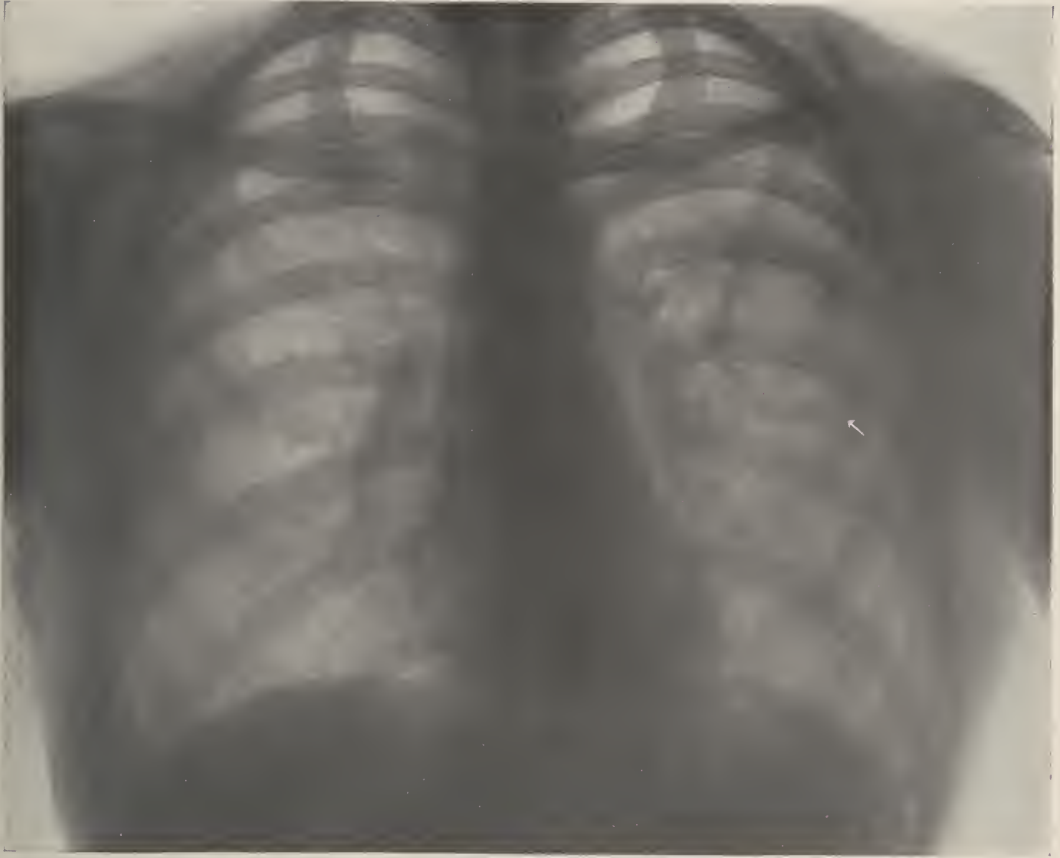


FIGURE 95

Cavity with incomplete wall in left upper lobe. Lung about cavity is not infiltrated. See figure 96.

in figure 96 to convince us of the presence of a true cavity with a shifting fluid level. In each of these cases, the presence of tubercle bacilli in the sputum and the subsequent development of an extensive tuberculous process, left no doubt of the true nature of the disease.

The diagnostic difficulties which face the clinician in these cases are inherent in the nature of the pathological process. The characteristic physical signs of cavities of moderate size are to a great extent dependent on the transmission of sounds generated in them through the medium of consolidated lung tissue. Where there is little or no infiltration of the lung about the cavity, physical signs of the latter may be absent. In neither of the cases above illustrated were there any signs in the chest to suggest the presence of cavities; in fact in one of them, figure 94, there were no abnormal physical signs of any kind.



FIGURE 96

Same case as figure 95, showing shift of fluid level in cavity.

It is of interest to note that the cavities in these cases are not located in the usual situation in the apex or infraclavicular region but rather in the lower part of the upper lobe. Apical cavities are usually an accompaniment of an indurative process with fibrosis, a type of reaction which is characteristic of tuberculosis at the apices. This tendency diminishes as we recede from the apex and one is tempted to believe that the absence of infiltration and fibrosis about the cavities we are considering may be due to the fact that they resulted from a softening of an acute pneumonic tuberculosis.

UNILATERAL ADVANCED TUBERCULOSIS

It is a rule to which there are few exceptions, that advanced tuberculosis is bilateral. Even in cases with a moderate involvement, both apices will commonly be found affected although usually to an unequal extent. It is therefore of interest to consider briefly a group



FIGURE 97

Extensive unilateral tuberculosis of many years' duration.

of cases in which for a long period the tuberculous process although extensive, confines itself to one lung. The clinical history is commonly that of a chronic pulmonary inflammation with profuse expectora-

tion in which tubercle bacilli may be found only with difficulty, together with the preservation of a fair degree of health. In these cases one may find a dense shadow which obscures one lung almost completely, the opposite lung remaining uninvolved. Often the thoracic deformity and the displacement of the heart and mediastinum indicate a chronic fibrosing form of the disease which affects also the pleura. In other cases again the infiltration may be in the nature of a pneumonic consolidation of a considerable portion of a lung in which there is a minimum of fibrosis. (Fig. 97.)

Finally there is a small group of cases in which the induration of the lung and pleura terminates in the formation of tuberculous bronchiectases which may affect one lung exclusively. The bronchial dilatations may involve an upper lobe as in figure 98 or as in figure 99 predominantly the lower lobe. In each of these cases there is seen a striking thoracic deformity and a displacement of the heart and mediastinum. The case illustrated in figure 99 was of many years' standing; toward its end, the patient who had considerable dyspnoea, presented evidence of cardiac weakness. The bronchiectases, which affected mainly the lower lobe were complicated by a chronic pleurisy with pleuro-pericardial adhesions. The heart is drawn to the left and its outline is obscured by pleural thickening. On fluoroscopic examination, in deep inspiration, a forcible systolic impulse was visible in the diaphragmatic region. This was evidently due to an adherent pericardium; clinically there was a corresponding Broadbent sign.

This group of cases has an additional interest because of the difficulty which is often experienced in distinguishing them from chronic non-tuberculous disease of the lung. These patients not infrequently remain in good health for a long period during which tubercle bacilli are not found in the sputum after repeated examination. For this reason they are apt to be regarded as tuberculous or nontuberculous by the examiner according to his personal bias until either the discovery of tubercle bacilli or some complication of the disease clears up the diagnosis.

THE PERMANENCE OF THE ROENTGEN SHADOWS IN TUBERCULOSIS

The fact that so many cases of tuberculosis, especially those in the incipient stage, become clinically cured, naturally suggests a query as to what becomes of the shadows seen on the plate. With the subsidence of the inflammatory process, are all the infiltrations absorbed and does the lung resume its normal appearance? An answer to this question must take into account both the extent of the disease and the nature of



FIGURE 98

Chronic fibroid tuberculosis of right upper lobe with multiple bronchiectatic cavities.
Displacement of mediastinum.



FIGURE 99

Chronic unilateral fibroid tuberculosis with multiple cavities. Deformity of chest and pleuro-pericardial adhesions.

the pathological process in the individual case. There is no doubt that a few small infiltrations at an apex may entirely disappear so that subsequent Roentgen examination will show no trace of them. This, however, usually requires considerable time, perhaps months or years, during which the shadows persist with remarkably little change in their size. As a rule, in the ordinary process of healing with the development of fibrosis, definite evidences of the previous disease remain on the plate. To these we have already referred in a previous section.

When tuberculosis has passed beyond the incipient stage, especially when caseation has already supervened, we may look upon the Roentgen shadows as more or less permanent. The disease may become arrested and the symptoms subside; yet, aside from the changes due to fibrosis and calcification, the shadows will persist.

Nevertheless there are notable exceptions to this rule, cases in which most extensive infiltrations are susceptible of complete resorption with a restitution of the integrity of the lung. That we may understand such an unusual occurrence, it is necessary to call to mind the nature of the inflammatory reaction which underlies an acute tuberculous process. It will be recalled that its characteristic product is a sero-cellular exudate in the alveoli, which occurs either alone as an extensive pneumonia or as a zone of exudation about a group of tubercles. Now, it is a matter of common observation that pneumonic exudates do not necessarily go on to caseation but that after a variable period they may undergo partial or complete absorption, depending on the virulence of the toxin. We may instance in this connection certain cases of tuberculosis which are perhaps more common than is suspected, which begin as a pneumonic process at the apex of the lung. Judging from the plate and from the extent of the physical signs one is led to conclude that the patient has an advanced tuberculosis. Yet after a lapse of several months the exudate may entirely resorb, leaving behind only a few fibrosing tubercles. We may thus observe in figure 100 the complete disappearance within six months of a pneumonic tuberculosis of the right upper lobe which began with high fever, cough and hemoptysis.



FIGURE 100

Tuberculous pneumonia of right upper lobe, showing its complete resorption with cure, one year later.

The tuberculous pneumonias which complicate the more advanced forms of tuberculosis supply us with the most striking examples of evanescent Roentgen shadows. These pneumonias are frequently the result of the overflow from an apical cavity, whose toxic secretions may involve a whole lobe in an exudative process. We may illustrate this occurrence by the following case:



FIGURE 101

Acute tuberculous pneumonia about a chronic apical cavity. See figure 102.

A young woman who had had occasional cough and hemoptyses for some time, became acutely ill. For two weeks she had all the signs of an acute respiratory infection. The temperature was continuously high and she brought up quantities of muco-purulent expectoration mixed with bright blood. Physical examination revealed marked dulness over the right upper lobe with amphoric breathing and a few rales. At the height of the disease the plate, figure 101, revealed a dense pneumonic shadow involving the lower half of the right upper lobe which partly concealed a large cavity.

After two weeks the fever subsided gradually and with it all her symptoms. At this time the pneumonic shadow had entirely disappeared, bringing to light a large fibrous-walled cavity. (Figure 102.)



FIGURE 102

Same case as figure 101 three weeks later, showing complete absorption of pneumonia. Shows fibrous-walled cavity and displacement of mediastinum.

In cases of this type the question may properly be raised whether such a pneumonic process is a specific tuberculous one or whether, in view of its rapid disappearance, it may not be an ordinary lobar pneumonia. Of course in the individual case, this question cannot be positively answered as ordinary pneumonias may occur in tuberculous patients. Nevertheless, clinical experience emphasizes the wisdom of regarding them as tuberculous, especially when, as in this case, there is such an obvious cause for their production, as a chronic cavity. In the case which we have cited, we must assume that the virulence of the organisms or their toxins, which were generated in the old apical cavity, were so attenuated that the reaction to which they gave rise was an exudative one, in the nature of a gelatinous pneumonia, which did not go on to necrosis.

A like phenomenon may be observed in an interesting group of cases which occur especially in children in whom such an acute reaction develops about tuberculous bronchial lymph nodes. These children may suffer with recurring attacks of acute respiratory disease at intervals of several months. During the attacks, pneumonic shadows are seen extending from the root of the lung oftenest on the right side. After several weeks these shadows, which must represent acute exudative reactions, entirely disappear. (Fig. 121.) It is not improbable in view of these observations that many of the acute febrile attacks which mark the course of chronic tuberculosis are due to outbreaks of acute tuberculous broncho-pneumonia, which may later disappear. As Fraenkel has shown, the toxic products of the tubercle bacillus are capable of producing such reactions in the lungs, which may resorb without going on to caseation.

A consideration of these cases will make the examiner hesitate to render a prognosis and cause him to reflect with bewilderment on the vagaries of this remarkable disease. An early lesion whose insignificant

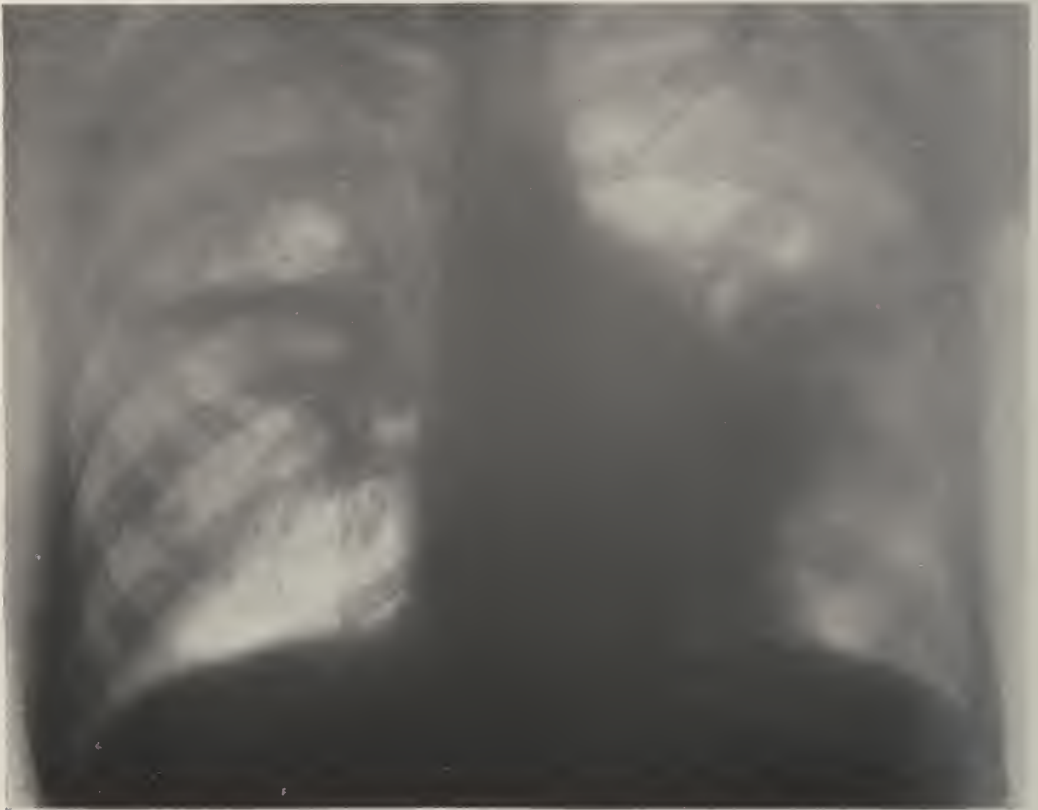


FIGURE 103

Acute caseous tuberculosis of both lungs with rapid breaking down and cavity formation. See figure 104.

size betrays him in an unguarded moment into a sanguine prognosis may go on uninterruptedly to a fatal ending, whereas a caseous pneumonia, so extensive as to discourage the confirmed optimist, may terminate most happily for the patient. Not only infiltrations but actual cavities, hollowed out of caseating lung may disappear in an unbelievably short time. Instance the following case whose outcome borders on the miraculous.

A woman of 38 years, suddenly overwhelmed by an acute tuberculous infection, presented the picture of extreme toxemia, high remittent fever and cough. During two weeks she lay at the point of death, which was momentarily expected, with exhausting sweats, rapid shallow breathing, extreme cyanosis and constant expectoration of purulent sputum, containing tubercle bacilli. Physical examination, borne out by the Roentgen plate, revealed extensive consolidation and rapid breaking down of the lungs, in which several large cavities had already formed. Rash indeed would have been any but a fatal prognosis. Yet, after three weeks, the temperature subsided and with a rapid improvement in well being the pulmonary infiltrations retrogressed and continued to almost complete resorption. (Figs. 103 and 104.)



FIGURE 104

Same case as figure 103 three weeks later. Absorption of most of the exudate in the lungs and disappearance of cavities. Subsequently, infiltration absorbed entirely and patient completely well.

THE ROENTGEN EXAMINATION IN CASES OF HEMOPTYSIS

The occurrence of hemoptysis justly creates a strong presumption of pulmonary tuberculosis in an individual so affected, unless there is some other obvious cause to explain it. This presumption is amply justified by clinical experience, with which the Roentgen findings are in complete accord. The Roentgen examination, by bringing to light foci of tuberculous disease in these cases has emphasized the frequent association of hemoptysis not only with advanced tuberculosis but also with incipient cases in which the infiltrations are too small to be recognized clinically.

We wish in this place to devote especial attention to the occasional cases of hemoptysis occurring in individuals who are otherwise well and in whom neither the physical examination nor the Roentgen plate bring to light any evidence of pulmonary tuberculosis. Such cases are very trying both to the patient and to the physician because, in spite of negative findings the disquieting suspicion remains that the patient harbors a small, undiscovered tuberculous focus.

The question arises under these circumstances whether we are justified in assuming that the hemoptysis is not tuberculous in origin. This query, which is so often propounded, merits a definite answer and if the Roentgenologist has confidence in his work, he will not hesitate to give it. It has been our experience that when a hemoptysis is due to tuberculosis of the lung, infiltrations can invariably be demonstrated on the Roentgen plate. Conversely, if such infiltrations cannot be found the hemoptysis has some other cause than pulmonary tuberculosis and it becomes imperative for the clinician to investigate the respiratory tract for other evidence of disease.

In our experience with these patients we have encountered a not inconsiderable number in which the spitting of blood was due to lesions in the respiratory tract other than tuberculosis. We may mention among others the cases of bronchial varix and congestion of the bronchial mucosa from ill understood causes. There are also cases of familial hemoptysis and others which show a purpuric tendency in which blood is expectorated without any pulmonary disease. Chronic cardiac disease, new growths, pulmonary syphilis and the various chronic non-tuberculous infections of the lung may be cited as causes of hemoptysis which the Roentgen Ray may be instrumental in disclosing. Finally, vascular sclerosis affecting the bronchial arteries, which may be associated with arterial hypertension, is also an occasional cause of severe hemoptysis. In this condition the Roentgen plate may reveal a hypertrophy of the left ventricle and thus throw indirect light on the cause of the hemoptysis.

Although vicarious menstruation is not as commonly invoked as a cause of hemoptysis as formerly and should not seriously be considered until the resources of physical examination have been exhausted, we have nevertheless encountered a few cases in which during the premenstrual period or during menstruation, hemoptysis, which could not be explained in any other way, has frequently recurred.

We have finally to consider the possible relationship between hemoptysis and tuberculosis of the bronchial lymph nodes. In the absence of any tuberculous infiltration in the lung the question arises whether the enlarged lymph nodes which are so frequently found at their roots, may by themselves be the cause of the spitting of blood. Bronchoscopic examination in these cases has at times revealed a congestion of the bronchial mucosa from which the bleeding occurred and to which, it is conceivable, the enlarged lymph nodes may have contributed. Yet when it is remembered that disease of the bronchial lymph nodes is very common, whereas hemoptysis in this type of case is extremely rare, it is not probable that there is a causal relationship between them.

Hemoptysis is a symptom of the greatest gravity and its importance always warrants a most painstaking examination of the lungs. Particularly in cases in which its cause is obscure does it demand most critical consideration and it is no exaggeration to say that here the Roentgen examination, especially in its negative aspects, is of transcendent value.

THE BEARING OF THE ROENTGEN FINDINGS ON THE QUESTION OF ACTIVITY

What information does the Roentgen plate afford us in regard to the activity of a tuberculous process? This query is constantly propounded by the clinician in the natural desire to enlighten himself as to the prognosis and treatment of his case. In resolving this question we can do no better than to consider for a moment what constitutes activity. Clinical activity is the result of certain changes in the tuberculous focus, by virtue of which a general bodily reaction is produced. It is important to realize that these changes are biochemical, not histological and that they cannot be inferred from the microscopic or macroscopic appearance of the lung. Thus, a tuberculous individual may at one time exhibit all the clinical signs of activity and a short time thereafter be free from them. Yet, as far as the eye or the microscope can determine the pathological process in his lungs may remain unaltered.

It is not to be expected therefore that the Roentgen plate, which is but a rough image of the actual deposits in the lungs, will be more informative than the microscope. In fact, there is no character of the Roentgen shadows of pulmonary tuberculosis from which either activ-

ity or quiescence of the lesions may be deduced. This statement finds its confirmation in the daily experience of the Roentgenologist both in incipient and advanced cases. A shadow which is discovered during the incipient stage of tuberculosis may and often does remain unchanged in appearance long after the signs and symptoms of activity have subsided. Even in the acute disseminated forms of the disease in which the appearance of the shadows leaves no doubt of the virulence of the infection, the Roentgen plate furnishes us no basis for assuming that the disease is an active one. It is true that such shadows bear witness to the acuteness of the process, which must therefore at some time have been active; yet this activity need not exist at the moment.

The same observations will apply to the healing and quiescence of tuberculous infiltrations. The presence of strands of fibrous tissue in the infiltrations will merely indicate that there is a tendency to healing, not that healing has actually been accomplished. The greater the amount of fibrosis, shrinking of the chest and displacement of the thoracic organs, the greater is the probability that the disease is no longer active. But it must be remembered that even a fibrosing process may be associated with activity and inferences as to actual quiescence must be made reservedly. In our analysis of the Roentgen plate we may appraise the contending forces which on the one hand make for activity and on the other hand strive for the arrest of the disease and though we may note a preponderance of one or the other, we cannot express our conclusions in terms of activity.

It would therefore mark an advance in the interpretation of plates if such terms as "active" and "quiescent" and similar adjectives, as descriptive of infiltrations, were discarded. As shadows, they have no character of activity or quiescence. It would be more in conformity with our knowledge to describe them in general terms, as, for example, "of recent" or "remote origin" or even as "acute" or "chronic." If a more exact pathological interpretation is desirable, such terms as caseous, exudative or fibroid may properly be applied to them.

THE BEARING OF THE ROENTGEN FINDINGS ON THE PATHOLOGY OF TUBERCULOSIS

We have sought, in the foregoing pages, so to interpret the Roentgen shadows of pulmonary tuberculosis, as to bring them into harmony, as far as this is possible, with recognized pathological types. In essaying such a correlation, we must however remember that the morbid changes in the lungs are so complicated that the conglomerate of shadows which we see on the plate can only roughly outline the underlying disease. The conservative Roentgenologist, bearing in mind the inherent limitations of his art will therefore refrain from reading too much into his plates and he will be content to make such general state-

ments in regard to the infiltrations as we have indicated. The more exact determination of the pathological or clinical type of the disease and especially the question of its activity, he will wisely leave to the clinician.

There are however, other aspects of this disease concerning which the thoughtful examiner is tempted to speculate. As the tuberculous infection in its manifold forms is revealed to him certain problems in regard to its pathogenesis, especially its original site in the lung and its mode of progression, will arouse his interest. We have in the Roentgen plate, a means of inquiry into these questions, which in some respects exceeds in value the autopsy itself. In no other way have we the opportunity, usually denied to the pathologist to observe the very beginnings of this disease and by successive examinations to study its gradual or rapid propagation throughout the lungs.

THE SITE OF THE INCIPIENT LESION

It is the common belief, amply supported by evidence from various sources that pulmonary tuberculosis in adults is a metastatic infection from a previously existing focus, which is usually located in the bronchial lymph nodes. For reasons not well understood the apical or sub-apical regions are especially predisposed to the disease, so that the tubercle bacilli are there deposited. The earliest lesion is therefore situated at a remote distance from the lymph nodes at the root of the lung whence the infection originated. This theory of the mechanism of pulmonary infection receives a striking confirmation from the Roentgen examination. On the plate the infiltrations of incipient tuberculosis are almost invariably located in the upper lobes near their periphery. The deposits are small and circumscribed and, what is of particular interest, that portion of the lung which lies between the diseased area and the hilum is uninvolved.

Opposed to this theory, a number of Roentgenologists have advanced a contrary one namely, that tuberculosis begins at the root of the lung whence it extends to the periphery. This theory of a primary hilum tuberculosis with a secondary pulmonary involvement early enlisted many adherents, who based it almost entirely on a reading of the plates, with little pathological or clinical support.

These opposing views have more than a theoretic interest. They are founded on fundamentally different interpretations of the Roentgen plates, both of which obviously cannot be correct. Such being the case, it is necessary to clear up the points at issue before the Roentgen interpretation of tuberculosis can achieve a firm and rational basis. We therefore propose to discuss them at greater length.

We shall first consider the evidence which has been adduced to support the theory of a central origin of the tuberculous process. The fact that this is contrary to accepted pathological doctrine is an added reason for examining this evidence critically.

In the early era of Roentgenology when it was realized that pathological deposits in the lungs could be recognized on the plate, enthusiastic observers, who for the first time saw the massive hilum shadows, promptly regarded them as evidence of tuberculosis. The fact that these shadows were occasionally associated with clinical signs of respiratory disease and the absence of infiltrations in the periphery of the lung, naturally strengthened this belief.

Having assumed that tuberculosis begins at the root of the lung, it was next necessary to explain the method whereby the outlying portions of the lungs are involved. This was conceived as an extension of the infection outward from the root, along the peribronchial lymphatics to the surface of the lung. The complete theory therefore involves two distinct suppositions. (1) A hilum tuberculosis, as the origin of the disease, and (2) its peribronchial centrifugal extension. It will be our task now to test the truth of the Roentgenological basis for these assumptions.

THE ROENTGEN EVIDENCE OF A PRIMARY HILUM TUBERCULOSIS

When we inquire into the evidence on which Roentgenologists have based their belief in a hilum tuberculosis, it will be found that this is of the slenderest and is founded on a misinterpretation of the root shadows. These shadows are cast by different structures, such as blood vessels, bronchi and lymph nodes, all of which are anatomically distinct from the lung parenchyma. An increase in the size of these structures from disease brings about a widening of the hilum shadows, which may extend for some distance into the pulmonary fields, without the lungs themselves being involved in the disease. In fact, there is every reason to believe, that in adults, an enlargement of the roots may be traced to the almost universal disease of the bronchial lymph nodes. Strictly speaking, there is no such thing as a normal hilum. Practically all individuals bear the marks of numerous respiratory infections, whether tuberculous or otherwise, which are visible in the bronchial lymph nodes. These exhibit, in varying degrees such diverse pathological changes as hyperplasia, anthracosis, calcification and caseation which are revealed on the plates as widened and irregular root shadows.

The advocates of the theory of primary hilum tuberculosis attach great importance to these shadows and in cases of suspected tuberculosis regard them as confirmatory of the diagnosis. And yet, everyday experience is strongly opposed to such a view. Enlarged and

diseased bronchial nodes are almost universal, whereas clinical symptoms resulting from them are relatively rare. Especially in respect to tuberculosis of these nodes, must we realize that although they are infected, the pathological process is usually in a state of quiescence and that symptoms arising from their activity are of rare occurrence. When such symptoms occur in patients who exhibit enlarged root shadows, it will be nearer the truth to explain them as the result of chronic bronchitis and emphysema or the various respiratory infections which are secondary to accessory sinus disease. To regard these shadows as evidence of an involvement of the lung root in a tuberculous infiltration is fatal to a rational interpretation of the plates; a large percentage of mankind would then be pronounced clinically tuberculous and the Roentgen diagnosis would be reduced to absurdity.

THE ROENTGEN EVIDENCE OF PERIBRONCHIAL TUBERCULOSIS

The Roentgen evidence in support of a peribronchial tuberculosis is derived from the coarse radiating strands which traverse the chest from the root to the periphery, whose irregular and at times nodular outlines seem to suggest a tuberculous infiltration about the bronchi. This assumption has made such an appeal to Roentgenologists, that peri-bronchial tuberculosis has for a long time had a prominent place in the diagnosis of this disease. And yet, it is undoubtedly founded on an error in interpretation which arises mainly from a misconception of the anatomical basis of these radiating shadows. There are ample grounds for believing that these shadows, even when they are much enlarged, have no connection with the bronchi at all, but represent a dilatation of the blood vessels and lymphatics of the lung and that therefore they cannot be regarded as evidence of a peribronchial or other form of tuberculosis. The reasons for this belief are the following:

(a) The pulmonary markings, which are predominantly the shadows of blood vessels, may become broader and irregular under various circumstances, when there can be no question of tuberculosis. Plates of healthy individuals, made in the prone position, especially when the tube is very close to the patient, exhibit coarse radiating shadows which are identical with those of so-called peribronchial tuberculosis. Aside from these external agencies, any obstruction to the return flow of blood from the lungs, will effect a widening of the lung arborizations and lead to a similar Roentgen appearance. The most striking example of this will be found in cases of advanced mitral disease, in which the pulmonary fields are traversed by irregular linear shadows of distended vessels which issue from a much enlarged hilum. (Figs. 4 and 5.) Were these shadows consistently interpreted in accordance with the peribronchial conception of tuberculosis, a large

majority of cardiac patients, would have to be considered tuberculous. This we know to be contrary to medical experience which teaches only the exceptional association of tuberculosis and heart disease.

(b) A similar mechanism will explain the exaggerated markings so often seen in adults with enlarged anthracotic or tuberculous bronchial nodes. Pressure of these nodes on the afferent vessels at the root of the lung and consequent engorgement of the pulmonary blood vessels and lymphatics offers a more rational explanation of these shadows, than does a peribronchial tuberculosis. Failure so to interpret them has in the past brought chaos to the Roentgen diagnosis of tuberculosis. Untold numbers of patients, with no more serious ailment than a chronic bronchitis or a quiescent glandular tuberculosis have been pronounced tuberculous because in the reading of their plates the exaggerated shadows of a normal component of the lung have been mistaken for disease.

(c) A more convincing proof of the vascular basis of these strands may be found in the plates of children. As is well known, tuberculosis in children is usually confined to the bronchial lymph nodes and in only a small proportion does it involve the parenchyma of the lung. Clinically, the most prominent symptoms of this adenopathy are due to pressure of these nodes at the roots and we may expect to find on the plate evidence of this pressure in an increased vascularity of the lungs. This, in fact, is the case. It is almost the rule, in cases of well marked glandular tuberculosis in children, to see coarse, nodular strands which extend outward into the lung from the roots, being most prominent in the paravertebral and infraclavicular regions. (Fig. 116.) From the frequency of their occurrence and the known variety of pulmonary involvement in children, we are forced to conclude that these shadows are not cast by tuberculous infiltrations but are rather blood vessels and lymphatics which are distended because of an interference with the return flow of blood and lymph from the lungs.

ROENTGEN EVIDENCE OF THE PERIPHERAL ORIGIN OF TUBERCULOSIS

The positive evidence in favor of a peripheral origin of tuberculosis has been set forth at length in the section devoted to incipient tuberculosis. We have there fully illustrated the characteristics of the early infiltrations, which do not show in their distribution, any relation to the course of the bronchi nor any connection with the hilum of the lung. In fact, in truly incipient cases, that portion of the lung situated between the infiltrations at the periphery and the hilum, is absolutely devoid of abnormal shadows, a condition which is in complete harmony with the theory of pulmonary infection here set forth.

We have dealt with this subject at some length because it is of crucial importance in the rational interpretation of the Roentgen plate. Beginners especially are too prone to attach significance to every shadow on the plate even though it be normal and to seek an explanation for it in terms of disease. We believe that only by restricting the Roentgen evidences of tuberculosis to definite pulmonary infiltrations, will the diagnosis achieve the decisive character to which it may justly aspire. We are further convinced that, far from circumscribing the scope of the Roentgen examination, such a restriction will extend it and enhance its value and will bring it into harmony with the coordinate branches of clinical medicine and pathology.

THE ROENTGEN EXAMINATION AND THE PHYSICAL SIGNS

When we compare the extent of the tuberculous process as seen on the plate with the physical signs, especially in respect to dulness, a marked discrepancy will frequently be noted. One will be amazed to see extensive shadows, involving a large part of the lungs in certain cases in which little or no dulness can be elicited. This is especially true of the lower lobes where emphysematous lung, overlying an area of consolidation, may mask the dulness. We must constantly bear in mind that the phenomena of percussion are purely acoustic and have no necessary correspondence with those of the Roentgen Ray, which are purely optical. The attempt to correlate them is therefore often attended with discordant results.

To an even lesser degree is there a correspondence between the appearance of the Roentgen shadow and the presence or absence of rales. An extensive consolidation due to pneumonic tuberculosis may in the one case be accompanied by an abundance of rales, whereas in another case with an identical Roentgen picture, rales may be absent.

It is however in the interpretation of incipient tuberculosis that an accordance between the plate and the physical signs, especially in respect to apical rales, is most eagerly sought. Are these rales necessarily an evidence of pulmonary tuberculosis? Do the Roentgen findings support such an assumption? We are confronted here by a problem of great moment in the solution of which the Roentgen examination is bound to play a prominent role.

Apical rales are justly invested in the mind of the physician with an ominous significance and experience proves a large percentage of them to be of tuberculous origin. With this the Roentgen examination is in entire accord. Nevertheless we encounter from time to time patients in whom despite the presence of more or less constant rales at an apex, the Roentgen plate shows no corresponding infiltrations. Shall these patients none the less be considered tuberculous? In a matter of such practical importance dogmatism is unbecoming both in the clinician and

the Roentgenologist. Neither can the apical rales be lightly dismissed by the one nor the evidence of the negative plate be disregarded by the other. In a doubtful case it will therefore be wise to keep the patient under observation. We wish however to emphasize that there are undoubtedly cases with apical rales which are not tuberculous and in which the negative Roentgen finding will encourage the physician to take a less serious view of the case.

What the pathological process may be which gives rise to these rales, it is difficult to say. Other respiratory infections besides tuberculosis can certainly induce an apical catarrh. The influence of apical atelectasis in the production of rales must also be kept in mind. Finally the scars of healed tuberculous lesions, with a retraction of the apices are a prolific source of apical rales which may persist for a considerable time after the cure of the disease. Although ultimately the decision as to whether an actual tuberculosis exists must depend on the clinical symptoms such as the temperature, pulse rate and sputum, it has been our practice to regard these cases, in which the Roentgen plate is persistently negative, as non-tuberculous. The wisdom of this attitude has almost invariably been confirmed by their subsequent course. These patients practically always recover promptly or some other cause for their respiratory symptoms is found.

THE RELATIVE VALUE OF THE ROENTGEN AND PHYSICAL EXAMINATION

It is to be deplored that even at the present time there is much difference of opinion as to the relative value of the physical and the Roentgen examination in the diagnosis of tuberculosis. It is often asked whether the Roentgen Ray represents an advance over the older clinical methods of percussion and auscultation. Is it possible by means of the Roentgen plate to discover infiltrations of incipient tuberculosis at a time when physical signs of the disease are not yet present?

The clinician who, as a rule, is unskilled in the use of the Roentgen Ray has been reluctant to concede its value for a number of reasons. In the first place he was possessed of an unwarranted fear that the Roentgen examination would overshadow or even supplant the clinical examination. Secondly, the shortcomings of the Roentgenologist himself, on whom the clinician has had to depend for the reading of the plates, have engendered a distrust for the Roentgen findings. The Roentgenologist has too often based his diagnosis on technically imperfect plates and he has consequently fallen into error. Ignorance of the anatomical basis of the normal lung shadows also led him to advance erroneous theories of the origin and mode of spread of the disease in the lungs which have completely vitiated his interpretations. These interpretations are moreover clothed in a picturesque phraseology which is both inaccurate and

confusing to the clinician. Finally there has been so little unanimity among Roentgenologists in the interpretation of the plates that the clinician has not been encouraged to depend on them.

The clinician however bases his preference for the physical examination on more positive grounds than the foregoing. Stated in its most uncompromising form, the belief of many specialists in physical diagnosis is that incipient tuberculosis cannot be determined by the Roentgen Ray or at least that it is sooner discovered by auscultation and percussion. We have here a simple issue of fact which is worthy of most careful consideration.

In the first place, it is proper to inquire whether the physical signs of incipient tuberculosis are of such a definite character that physicians as a rule find themselves in agreement in its diagnosis. Now as a matter of fact this is far from the case. Assuming that a patient has such equivocal signs at an apex as diminished or granular breathing and slight dulness, different examiners will not put the same interpretation on these changes. The conservative physician will demand more positive evidence upon which to diagnose tuberculosis and will hold his opinion in abeyance pending the result of sputum examinations and other clinical tests. There is however a school of clinicians among whom are numbered many specialists in this disease, who unhesitatingly base a diagnosis of tuberculosis on slight abnormalities in breathing associated with more or less constant apical rales.

To those who hold the physical examination in such high regard, the Roentgen plate with its frequent negative finding, will of course, be disappointing. The clinical extremist, confident of his skill and fortified by his personal bias will at once conclude that the Roentgen plate is of little value. Yet we venture the opinion that the importance of slight breathing changes and apical rales has been altogether exaggerated and that in many cases such changes have been due to catarrhal processes other than tuberculosis. It would make an instructive commentary on the physical examination were it possible to estimate the number of patients who in the past have mistakenly been pronounced tuberculous because of slight apical changes. It is not the least of the achievements of the Roentgen Ray that in these doubtful cases the negative Roentgen finding may afford the conservative examiner the assurance that his patient is not tuberculous.

It is our belief, which daily experience is strengthening, that in the vast majority of cases of so-called incipient tuberculosis, definite infiltrations can be demonstrated by a well executed Roentgen examination. The converse of this is equally true, namely that a negative Roentgen plate effectually excludes tuberculosis. We wish to emphasize, however, that by infiltrations are meant the definite shadows which we

have described as characteristic of tuberculosis, not the so-called peribronchial infiltrations and root shadows, which are so often, to the detriment of the patient, regarded as tuberculous.

We are moreover firmly convinced, that the Roentgen examination, when properly performed, is the most certain method for the early diagnosis of pulmonary tuberculosis and that by means of it the disease can usually be discovered previous to the development of physical signs. Increasing experience is strengthening the impression that in many cases in which distinct physical signs are present, the tuberculous process has already passed beyond the incipient stage, as is shown by the extent of the shadows on the plate. We have had occasion to point out in our discussion of beginning tuberculosis how frequently the deposits are situated in the infraclavicular region. Here the Roentgen plate enjoys a pronounced advantage over the physical examination because the signs are hard to elicit. When rales are finally heard it is often found that the process has already extended beyond the incipient stage.

The superiority of the Roentgen Ray in the detection of the advanced lesions of tuberculosis is contested by scarcely any one today. It is an almost invariable experience that the plate discloses a much larger area of infiltration than the physical examination leads us to expect. This is increasingly true the further we recede from the apices and approach the bases where the infiltrations may be deeply seated. An extensive tuberculous broncho-pneumonia in a lower lobe may thus be entirely overlooked because of a surrounding compensatory **emphysema** which will mask both dulness and breathing changes. The physical examination here relies mainly on rales, which may be inconstant and which are, roughly, an evidence of activity. The Roentgen Ray on the other hand, unearths infiltrations, which are an expression of the total tuberculous process, active and inactive.

Particularly in chronic and quiescent cases, the extensive shadows to be seen on the plate may be out of all proportion to the meager physical signs. The diversity of pathological changes such as calcification, caseous broncho-pneumonia, miliary infiltrations, extensive fibrosis with cavitation and displacement of the thoracic viscera, are faithfully mirrored forth on the plate where they supply a wealth of information which the physical examination can never hope to rival.

It should not be presumed, from this catalog of the merits of the Roentgen examination that it is all-sufficient for purposes of diagnosis and that therefore the physical examination can be dispensed with. A Roentgen plate cannot take the place of a careful clinical examination; to attempt this would be to pervert its function. Nothing can replace the periodic percussion and auscultation of the chest for evidence of

activity or quiescence of the lesion or of its extension. The Roentgen examination must be utilized by the clinician as an adjunct to his other methods in order to achieve their greatest usefulness. When it is so used, it is safe to say that few clinicians at the present day will be content to forego the great help which the Roentgen Ray can afford them.

CHAPTER VI

Pulmonary Tuberculosis in Children

The tuberculous infection manifests itself so differently in infants and children from adults that it is desirable to treat its Roentgen diagnosis in a special chapter.

In recent years our knowledge of tuberculosis of the lungs in children has been extended in various directions. This we owe to the pathological studies of Albrecht and Ghon, to the extensive application of the various immunity reactions to the living child especially the von Pirquet test and finally to the correlation of clinical, laboratory and autopsy findings which led to the illuminating conception of the disease advanced by Hamburger. Further, the obscure and difficult problems involved in the origin and mode of dissemination of tuberculosis in the lungs have engaged the experimental pathologists, notably Bacmeister, the results of whose experiments have helped to explain the mechanism of human infection.

Until recently, the clinical diagnosis of tuberculosis in children had not kept abreast of the brilliant strides of pathology and immunology. Its progress was stayed by the difficulty of accurately determining the site, extent and type of tuberculous lesions which were present in the individual case, a difficulty which is far greater in infants and children than it is in adults. It is a peculiarity of tuberculosis in children that in its most common forms it occasions but few physical signs and these are difficult to elicit. It is essentially a disease of symptoms in the interpretation of which the physical examination too often fails.

Happily, the Roentgen-Ray has brought to the diagnosis a method of great exactitude, whose application is not subject to the limitations of the older methods. It penetrates the remotest recesses of the chest and regardless of the absence of physical signs, reveals in the one case the very beginnings of the disease and in the other its most advanced forms.

It will be desirable to rehearse briefly the pathology of tuberculosis particularly as it refers to the child, in order that we may interpret intelligently the data furnished by the Roentgen examination.

Perhaps the most useful conception in relation to this subject we owe to Hamburger. He has pointed out the marked similarity in the sequence of their manifestations, between tuberculosis and syphilis. Just as in syphilis, there is a primary, secondary and tertiary stage, each characterized by distinct kinds of inflammatory reaction, so in tuberculosis we have a chronic infection, beginning nearly always in childhood

which, in its evolution in the body assumes various forms. Here also we can recognize a primary stage or initial infection which is shortly succeeded by a secondary stage, to be followed after a longer interval by a tertiary tuberculosis. Just as the tertiary form of syphilis or gumma does not occur at the time of the initial lesion, so with rare exceptions the tertiary or adult form of tuberculosis does not occur in childhood, the period of initial infection.

Let us now observe the sequence of events which follow the entrance of the tubercle bacillus into the body. Be this by way of the bronchial tubes by inhalation or through the blood or lymphatics from a focus elsewhere in the body, the primary lesion of the disease is practically always an agglomeration of tubercles within the lung parenchyma, which varies in size roughly from that of a pea to a cherry. Most often it is found in the anterior part of the upper lobe just beneath the pleura and it is often accompanied by changes in the latter.

So ubiquitous is this disease, that up to the age of puberty, over 90% of city dwelling children have passed through such a period of pulmonary infection. Since a majority of the children recover from this primitive infection, the initial lesion being of small size, it is fair to assume that the number of invading organisms is small or their virulence attenuated. In cases of presumably massive infection, especially when this occurs in infants, the disease may be a very virulent one and result in early death. The initial focus may then be of great extent and present the clinical picture of infantile phthisis.

The second period of the disease follows closely on the primary infection. During it there is a tuberculous enlargement of the regional bronchial lymph nodes which remains after the pulmonary focus has completely healed. Disease of these nodes, situated at the roots of the lungs with the manifold symptoms and signs resulting from their tumefaction, furnishes the dominant features of the tuberculous infection in childhood.

Of greater importance however than merely local changes in the chest are the subtle reactions of the tissues to the tubercle bacillus which result on the one hand in a condition of allergy, as evidenced by the response to tuberculin injections and on the other hand in a partial immunity to further infection. The significance of the latter must be appreciated for a proper understanding of the evolution of tuberculosis in children. The existence of the initial lesion, with the continued absorption from it of small amounts of tubercle toxin, apparently so affects the reactivity of the lungs that their response to subsequent infection by the tubercle bacillus is notably modified. In the first place the child is shielded from massive infection and dissemination of the disease by the resistance it has acquired; secondly, the type of reaction in the lungs to later infection is changed from the acute, exudative and

caseous, to the more chronic, indurative form. This indurative form of tuberculosis, which we may speak of as its tertiary stage, is practically confined to adult life or later childhood. It is usually apical in situation, has a great tendency to fibrosis and is regarded as the unique reaction of a lung, which has become habituated to the tuberculous virus by previous and long standing infection. It always presupposes a primary focus in the lung which has usually existed from early childhood.

We may then summarize the events which follow the entrance of the tubercle bacillus into the child's lungs as follows. If the attack is a massive or virulent one, the child is overcome by an acute, caseous and often rapidly fatal disease. If the invasion is mild, there results a primary lesion of small extent, situated anywhere in the lung, which usually goes on to cure by fibrosis or calcification. Coincidentally there is a tuberculous adenopathy which may remain latent or may manifest itself clinically some time after the primary focus has healed. Finally, toward puberty, or more commonly in early adult life, there may be a tertiary infection of the lung from the mobilization of tubercle bacilli in the bronchial nodes or by massive infection from without. This tertiary form of the disease which is found nearly always in the upper lobes, because of the altered reactivity of the lung, is a chronic indurative one and is characteristic of the disease in adults; its further course has already been discussed in the chapter on adult tuberculosis.

We shall now proceed to the interpretation of the Roentgen findings and we shall attempt to apply to them, as far as we may, the foregoing considerations.

With respect to the classification of the various stages of the disease, we will find that the division into the conventional first, second and third stages is inapplicable to children, because the infection does not progress, as it does in adults, from the apex to the base. We shall therefore depart from it entirely and, unhampered by the uncertainties of physical signs, base our description of the different forms of the disease on the appearance and distribution of the infiltrations as seen on the plates. The various types of tuberculosis in infants and children will then be found to conform to the following grouping:

- I. Primary Tuberculosis:
 - (a) Focal primary lesion.
 - (b) Caseous pneumonia; infantile phthisis.
- II. Tuberculosis of the Intrathoracic lymph nodes.
- III. Hilum Tuberculosis.
- IV. Recurrent Hilum pneumonia.
- V. Miliary Tuberculosis.
- VI. Apical Tuberculosis and other adult forms.

PRIMARY TUBERCULOSIS

(a) **FOCAL PRIMARY LESION.** The initial lesion of tuberculosis practically always passes unnoticed. Its small size renders its recognition by physical examination impossible and its symptoms are probably mistaken for those of the numerous respiratory or other infections which are so prevalent in the early years of life. The Roentgenologist rarely has the opportunity to observe these cases in their acute stage, especially as they usually go on to rapid healing. Only in rare cases will he be fortunate to discover them while they are still active. He will then find on the plate a pneumonic infiltration of moderate extent, in any one of the lobes. Thus in figure 105 an early tuberculous process, pneumonic in



FIGURE 105

Primary tuberculosis in right lower lobe in 11 year old child. Enlargement of bronchial and paratracheal nodes.

type, is seen in the right lower lobe, in a girl eleven years of age. She had no previous pulmonary disease and her infection could be traced to a tuberculous mother who had died recently. It was sudden in onset and virulent, causing high remittent fever and cough. A bronchial

adenopathy, unusually marked for a child of this age was present and confirmed the probability of recent infection. After two months of acute illness, the symptoms subsided and the child was apparently well. The plate then showed a resorption of the initial focus, only a thickening of the interlobar fissure remaining. There was, however, a notable increase in the size of the bronchial lymph nodes. Again, in figure 106, the initial lesion occupies the region of the right hilum

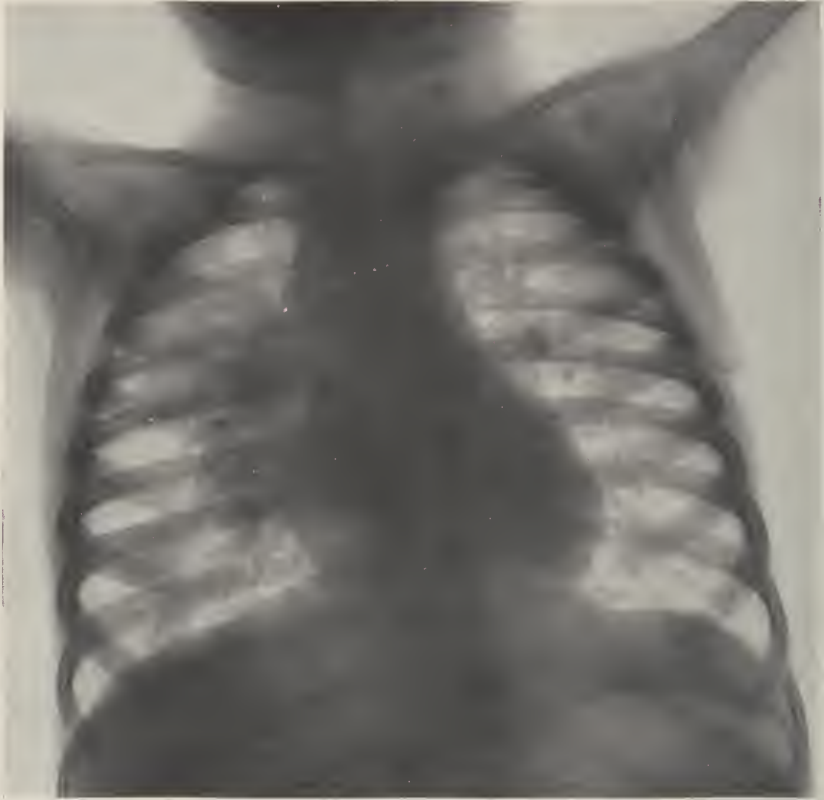


FIGURE 106

Primary tuberculosis in infant. The process consists of an infiltration at the root of the right lung. Bronchial nodes are much enlarged.

where it is in close proximity to large masses of tuberculous nodes. In this case, the extreme youth of the child, who was only a few months old, renders it probable that the pulmonary infiltration represented a primary rather than a secondary invasion of the lung.

More often, the Roentgenologist will find only the healed remains of the primitive infiltration, incidentally to the examination of the lungs for other forms of disease. While it is true in the larger number of cases that the primary lesion, having undergone fibrosis or other

form of healing, is no longer visible on the plate because of its small size, there are nevertheless numerous instances in which small discrete shadows are found on the plate, which can be nothing but the scars of past infection. As in figure 107, we then see small shadows, oftenest in



FIGURE 107

Calcified primary lesion in right upper lobe. - Calcification of bronchial nodes.

the upper lobes whose density and sharp outline indicate that they are the fibrous or calcified remnants of a primary tuberculous focus. In figure 108 the deposits are multiple and are found in both upper and



FIGURE 108

Multiple healed lesions of primary tuberculosis in left lung.

lower lobes. In figure 109 is illustrated a different type of healed lesion consisting of a strand of fibrous tissue in the left upper lobe. These healed lesions, whose pathogenesis presents so many points of interest, have no clinical importance. They are the cause of no symptoms on the part of the child, whose complaints can more often be referred to the enlarged, tuberculous lymph nodes whose shadows are a prominent feature of the Roentgen plates.



FIGURE 109

Fibrosis of primary lesion in left upper lobe. Tuberculous bronchial nodes.

The pleura over the site of the initial lesion is often involved in the inflammatory process. The changes are so slight however that except in the region of the interlobar fissure, they escape detection. In this region, a thickening of the pleura is demonstrable as a sharp linear shadow which traverses the chest from mid-line to axilla at the level of the fourth rib anteriorly. Routine study of the plates of children will reveal this shadow in a considerable number and it may be regarded in most cases as evidence of previous tuberculous infection in its vicinity.

(b) CASEOUS PNEUMONIA. INFANTILE PHTHISIS. Before pathologic studies revealed the frequency of pulmonary infection beginning in the early months of life and increasing year by year, tuberculosis in infancy was regarded as almost invariably fatal. We now know that even in infancy a tuberculous lesion can and frequently does completely heal. It remains true however, that the susceptibility to massive infection is very great in infancy and it may terminate in a most extensive caseous consolidation and necrosis of the lung, to which the term "infantile phthisis" is applied. Whether these extensive infiltrations are the result of the primary infection or whether they are secondary is not deducible from the Roentgen plate. They are in some case at least due to the rupture of a caseous lymph node into a bronchus with consequent bronchogenous dissemination of the disease. The type of infiltration seen in these cases is shown in figure 110 in which there is a caseous pneumonia of the right upper lobe with cavity formation. As is often the case with these advanced forms of infantile tuberculosis, other organs besides the lungs were affected. On the plate there may thus be seen a tuberculous abscess about the lower dorsal spine, which



FIGURE 110

Infantile phthisis. Caseous pneumonia of right upper lobe with cavitation. Tuberculous abscess about lower dorsal spine. (arrow.)

casts a pyriform shadow in the mid-line just above the diaphragm. The virulence of the infection in these cases is evidenced by the acute pneumonic type of reaction in the lung which rapidly terminates in cavity formation. (Fig. 111.)



FIGURE 111

Caseous pneumonia with multiple small cavities in an infant.

TUBERCULOSIS OF THE INTRATHORACIC LYMPH NODES

Disease of the intrathoracic lymph nodes is by far the most common clinical manifestation of tuberculosis in children. Investigation has conclusively shown that involvement of these nodes is invariably a sequel of the primary lesion in the lung. Be the latter healed or in a condition of caseation and softening, certain groups of nodes will be found enlarged and caseous or calcareous, their situation conforming to the law of regional node involvement. Owing to the greater frequency of primary disease in the right upper lobe we will often find the greatest degree of enlargement in the upper and lower tracheo-bronchial chain on the right side and also in the upper broncho-pulmonary nodes. The communication by means of lymphatics across the median line however accounts for the frequent glandular enlargement on both sides, even in cases of unilateral pulmonary disease.

It is a noteworthy fact that the glandular enlargement, especially in infants, is often out of all proportion to the size of the pulmonary lesion; a small caseous focus in the lung, draining through the pulmonary

or pleural lymphatics may thus be the cause of a most extensive cheesy tuberculosis of the nodes at the hilum, which in size greatly over-shadow the pulmonary lesion. This may be attributed to the wide lymphatics of infants which facilitate free drainage.

When we consider the clinical aspects of tuberculosis of the lymph nodes, we are impressed by the meagerness of the physical signs which they produce. It is essentially a disease of symptoms rather than of physical signs. The questionable value of the latter may be gauged by the number of alleged signs which various observers have described as characteristic, such as bronchial breathing over the manubrium (Heubner), Petrushky's spinalgia, dilatation of the thoracic veins, Koranyi's dullness below the fifth dorsal vertebra and finally D'Espine's sign. Some of these are present in individual cases and they are independent probably on a considerable enlargement of certain groups of nodes which are favorably situated for the production of the particular physical sign in question. Greater reliance is placed by the clinician on symptoms due to pressure on the bronchi, such as cough, dyspnoea and occasionally, a more or less characteristic stridor.



FIGURE 112

Tuberculous adenopathy in infant, unilateral. Enlargement of upper and lower broncho-pulmonary nodes on right side.

On the other hand, when we study the Roentgen plate for evidences of glandular tuberculosis, the great advance in diagnosis afforded by this method of examination is at once apparent. The diseased lymph nodes in all their variety of size, shape and situation are projected on the plate in dense shadow masses which extend beyond the mediastinum into the pulmonary fields.

We shall first discuss the tuberculous adenopathy of infants in whom it assumes a unique form. In them, the enlargement of the lymph nodes is relatively much greater than in older children, so much so, that the glandular mass may have the dimensions of a neoplasm. The first nodes which bear the brunt of the infection are the broncho-pulmonary groups, which lie within the lung fields close to the border of the heart and also the upper tracheo-bronchial group. The lower tracheo-bronchial or so-called bifurcation nodes are centrally situated and, being obscured by the heart, are not visible. Inability to see these nodes on the plate does not however limit the value of the Roentgen examination because their involvement is always associated with a tumefaction of the lower or upper broncho-pulmonary groups.

We may now illustrate the various Roentgen types which result from disease of different groups of nodes. In figure 112 we see an enlargement of the upper right group of nodes which have produced



FIGURE 113
Tuberculous adenopathy in infant, bilateral.

a one-sided widening of the mediastinum. It will be noted that the border of the shadow mass is convex and smooth and that the shadow is of uniform density. Usually, as in figure 113 the nodes on both sides are enlarged, the result being a marked, though irregular widening of the mediastinum. We may explain the homogeneity of the shadows by the pathological change in the nodes, which are often in a condition of complete caseation and are matted together into a solid mass. A rather common Roentgen picture of glandular disease in infants is shown in figure 114 where there is an almost uniform widening of the medi-



FIGURE 114

Tuberculous adenopathy in young child, simulating thymic enlargement.

astinum whose borders are sharply defined. The association of stridor and dyspnoea with such a shadow has frequently led to a diagnosis of enlarged thymus, which the autopsy almost invariably fails to confirm.

Tuberculosis of the paratracheal group of nodes, which attain their greatest size on the right side, has a special interest for us in view of its symptomatology. Attention has been called to a remarkable dyspnoea and stridor in infants which may be caused by the pressure of this chain of nodes on the lower trachea, when they become enlarged. On the plate they cast a dense elliptical shadow which extends from

the sterno-clavicular articulation to the eparterial bronchus. (Fig. 128.) They are as a rule associated with an enlargement of other bronchial lymph nodes and when they occur at the early age of two or six months usually have a bad prognosis.

Although the primary lesion in the lung which is responsible for the glandular swelling is occasionally to be seen on the plate, as a rule it is too small to be recognized. In fact, it is often with the greatest difficulty that it is found at autopsy after careful dissection of the lung. The relation which the massive consolidations and the miliary and conglomerate infiltrations that are occasionally seen on the plates of infants, bear to the tuberculous nodes, cannot always be defined from the Roentgen plate alone. While it is true that the pulmonary process may here be the primary one, it may on the contrary be a secondary infection due to the transport of bacilli through the circulation. For example, in figure 115 the enormous bilateral enlargement of the lymph nodes undoubtedly antedated the extensive lobular tuberculosis which was presumably metastatic in origin. Furthermore, some of the cases of



FIGURE 115

Massive enlargement of the bronchial nodes in an infant. Disseminated lobular tuberculosis.

infantile phthisis, with caseous pneumonia and cavitation are without doubt caused by the rupture of a cheesy lymph node into bronchus. In these cases the mechanism of the pulmonary affection can only be surmised but not proven from the Roentgen plate.

The extreme glandular enlargement in infants which we have described is justly invested with an ominous import, as the disease is commonly fatal. Yet there are striking exceptions which make it unwise to utter a hopeless prognosis. Within a few months or a year there may be an almost complete recession of the greatly enlarged nodes. In some degree this decrease in size is only apparent because with the rapid growth of the chest and of the thoracic viscera during the first year of life the lymph nodes become relatively smaller and lose in prominence.

There can be no doubt as to the tuberculous nature of this extreme glandular enlargement in infants. Other diseases in which similar shadows are found, such as mediastinal tumors, Hodgkin's disease and leukemia are of rare occurrence in infants. Furthermore the uniform presence of a positive tuberculin reaction, whose value in infants is decisive, testifies to their tuberculous basis.

The diagnosis of tuberculosis of the lymph nodes in older children raises a problem which has for a long time vexed both clinician and Roentgenologist. A decade ago, the Roentgenologist, carried away by enthusiasm for a new art, ascribed to the shadows of enlarged lymph nodes at the roots of the lungs an importance which sober afterthought has not justified. Since we have learned from extensive pathological studies and correlated tuberculin tests that tuberculization of over 90% of urban children has occurred by the time puberty is reached, it need occasion no surprise that the Roentgen plate almost invariably reveals shadows at the root of the lungs, which are presumably due to glandular tuberculosis. We must however discriminate between tuberculous infection, whose existence under the circumstances may be taken for granted and clinical disease, which will depend on the activity of the process in the lymph nodes. In regard to its activity, the Roentgen plate affords us no information.

In view of these facts the scope of the Roentgen Ray in the clinical diagnosis of tuberculous bronchial lymph nodes is much circumscribed. When the Roentgenologist affirms that these nodes are diseased his statement is of little value to the physician, because in most cases tuberculous glandular disease may be presumed to exist. The physician is more interested to know whether there is any causative relation between the enlarged nodes and the patient's symptoms. This can only be determined by the clinical and not by the Roentgen examination. This unfortunately is not always an easy matter. There are numerous

respiratory infections of non-tuberculous origin to which children are subject, which may give rise to cough, fever and swelling of the bronchial nodes. Before these symptoms can be attributed to tuberculosis it is necessary therefore to exclude such other causes as adenoids and paranasal infections and it may require a long period of observation before a decision is arrived at. It must moreover be remembered that it is not easy to distinguish on the plate between the moderate tumefaction of hyperplastic lymph nodes and the enlargement of tuberculous nodes, especially if caseation and calcification have not taken place.



FIGURE 116

Tuberculosis of bronchial lymph nodes. Note shadows of engorged vessels in upper lobes, often misinterpreted as "peribronchial" tuberculosis.

Tuberculous nodes in older children rarely attain the dimensions of those we have described in infants. With rare exceptions, (fig. 128), there is only a moderate increase in the extent and density of the root shadows which is due almost entirely to a swelling of the bronchopulmonary nodes. As a rule the enlargement is greater on one side than on the other and usually the dense well outlined shadows resulting from caseation are visible. (Figs. 116 and 117.) The irregular and at times



FIGURE 117

Tuberculous enlargement of right paratracheal nodes.

bizarre shadows of calcified nodes are seen only in older children and are practically never found in infants. (Fig. 118.)

It is worth while to emphasize once more the precautions which must be observed in the interpretation of certain pulmonary shadows in cases of bronchial node disease. When these nodes are so situated as to press on the effluent pulmonary vessels and lymphatics, the latter become over-distended with blood and lymph. The influence of this vascular congestion on the pulmonary markings is similar to that

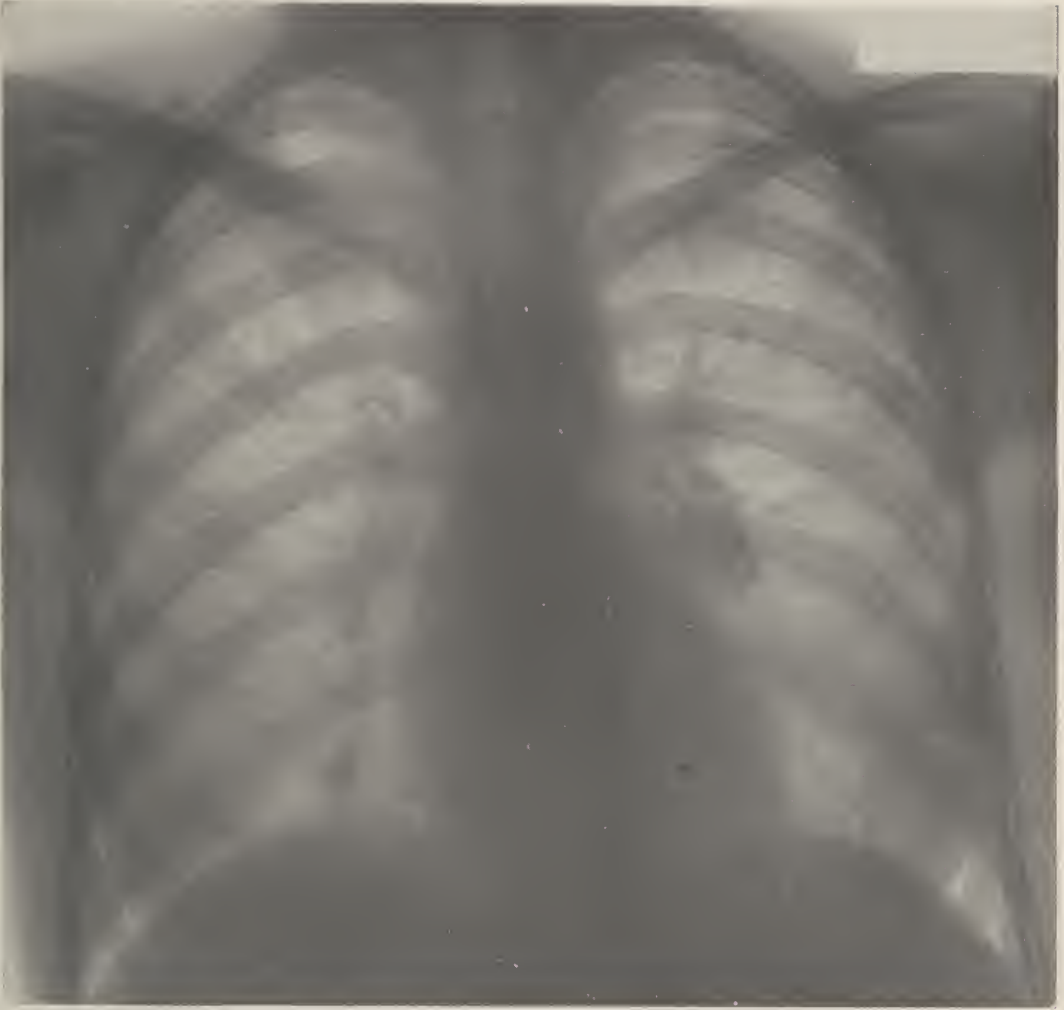


FIGURE 118

Calcification of left broncho-pulmonary nodes in an older child. Healed primary lesion at right base. See figure 127.

observed in cases of chronic valvular disease. The markings become broader and form a coarse network corresponding to the ramifications of the blood vessels and lymphatics, which is most prominent in the upper lobes. (Figs. 116 and 119.)

These shadows are often erroneously interpreted as a tuberculosis of the lungs spreading outward from the diseased lymph nodes, an interpretation which is calculated to exaggerate the seriousness of an otherwise mild infection. It also does violence to our conception of the

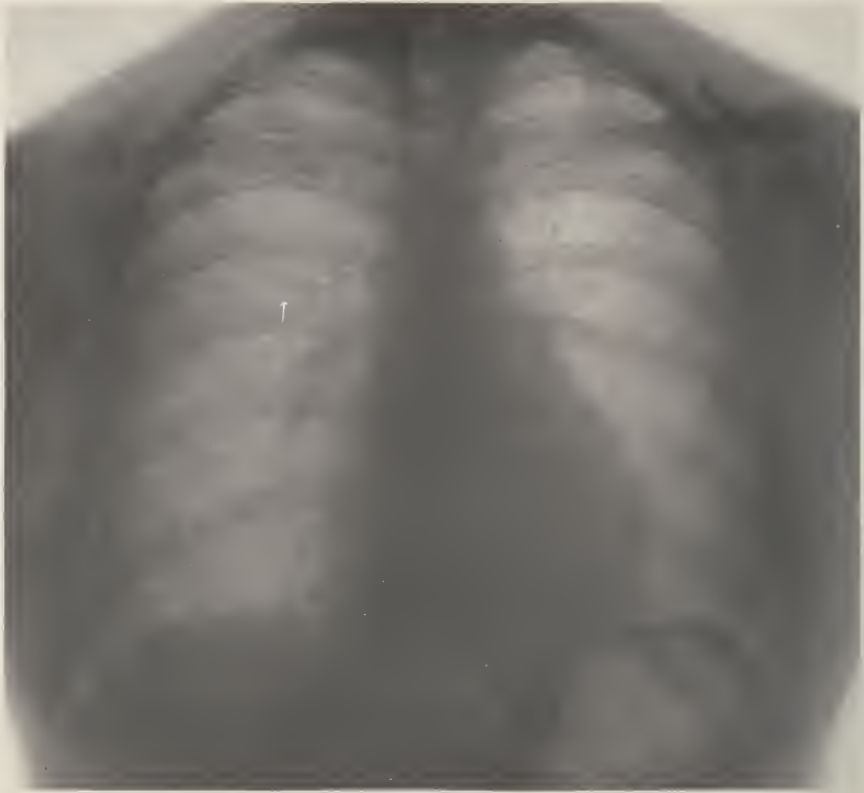


FIGURE 119

Vascular network in right upper lobe produced by pressure of lymph nodes at root of lung.

pathology of tuberculosis in children which, as we know, does not invade the lung in this manner but practically always remains confined to the lymph nodes. These shadows in themselves are not an evidence of pulmonary disease but are only a secondary result of the enlarged nodes and they have no significance.

When plates are interpreted with due regard to such sources of error, the clinician is apt to be disappointed at the limited positive value of the plate in the diagnosis of glandular tuberculosis. He will soon find that there is no correspondence between the size of the lymph nodes and the severity of the clinical symptoms. Nevertheless, the value of the Roentgen examination in these cases is surpassing, although in a negative sense. In cases of suspected tuberculosis in a child it is of paramount importance to determine whether there is actual pulmonary disease or whether the infection is confined to the bronchial nodes. Infiltration of the lung itself adds immeasurably to the gravity of the case,

whereas glandular tuberculosis is a relatively benign affection which in a majority of the cases becomes quiescent. It is because the diagnosis of pulmonary lesions in children, especially the relatively frequent infiltrations at the roots, is so uncertain by the ordinary methods of physical examination, that it becomes important whenever possible to exclude them by other means. For this reason the Roentgen plate has a major function in establishing an accurate diagnosis and therefore in providing a firm basis for prognosis.

HILUM TUBERCULOSIS

Tuberculosis of the hilum of the lung is a more common form of the disease in children than in adults. Aside from the interest which its usual situation arouses, it commands our attention because we owe



FIGURE 120

Hilum tuberculosis involving the middle lobe.

our knowledge of it as a clinical entity mainly to the Roentgen ray, by means of which alone, it is usually discovered. As its name implies, the tuberculous process originates at the root of the lung, from which it may extend outward a variable distance.

Two types of hilum tuberculosis must be distinguished inasmuch as they differ in their pathology and prognosis. The first occurs commonly in infants below one year of age and consists in a cheesy tuberculosis of the root tissues of the lung. It is essentially a caseous primary lesion which is rapidly progressive and has a bad prognosis. (Fig. 106.) Aside from the clinical symptoms which will serve to distinguish these cases from non-tuberculous pneumonia, the Roentgen examination reveals a difference in the configuration of their respective shadows. The pneumonic triangle usually has its base at the periphery, whereas in tuberculosis it is at the root.

A second variety of hilum tuberculosis occurs in later childhood, being most common at the age of six. It is clinically a milder infection than the infantile form and appears to have a relatively good prognosis. In its spread from the root of the lung, it may involve the upper, middle or lower lobes or the contiguous portions of all of them. Figure 120 illustrates in characteristic form a hilum tuberculosis invading the middle lobe, which was of some months' duration and presented the clinical picture of a subacute tuberculous pneumonia.

RECURRENT HILUM PNEUMONIA

There remains to be described a very interesting variety of hilum tuberculosis which is characterized by the recurrence of symptoms of pulmonary infection at intervals of weeks or months, during which there are cough, fever and at times a few physical signs, which are commonly audible anteriorly. During these attacks there will be seen on the Roentgen plate a triangular shadow whose base is at the root and is sharply outlined below by the interlobar fissure. After a lapse of several weeks the symptoms will subside and the Roentgen examination will show a complete disappearance of the shadow although a thickening of the fissure may remain. Such a cycle of events may occur every few months. In the case illustrated in figure 121 we were able to observe the appearance and resolution of the pneumonia on three occasions within a few months, associated each time with cough and fever and slight dulness, with a few rales over the right upper lobe. There can be little doubt that these recurrent infiltrations are tuberculous in origin. That they are not due to the ordinary pneumonic bacteria is probable from the mildness of the symptoms, which do not even cause the patients to take to bed. On the other hand the evanescent character of the exudate, which may disappear in a week, makes it necessary to assume that the inflammatory reaction is an exudative one which is susceptible of rapid



FIGURE 121

Recurrent hilum tuberculosis involving right upper lobe. Plate (a) represents the active stage of the disease. Plate (b) taken during interval, shows complete absorption of infiltration.

absorption without undergoing caseation. If we may speculate as to their nature we may conceive these hilum pneumonias as mild reactions in the vicinity of tuberculous lymph nodes which are similar to the exudative processes that are often found about recent lesions in the lungs of adults.

The difficulties in the physical diagnosis of hilum tuberculosis are in striking contrast to the ease of its demonstration by the Roentgen Ray. The infiltration is so deeply seated at the root of the lung that few if any physical signs are transmitted to the ear and the belief of the clinician is severely tested when, in a case with no physical signs, he is informed that an extensive infiltration nevertheless exists. In respect to the symptoms, these are often mild and differ little, if at all, from those which result from tuberculous lymph nodes. It is evident that in the diagnosis of hilum tuberculosis and especially in its differentiation from uncomplicated tuberculosis of the bronchial nodes, we are almost entirely dependent on the Roentgen examination.

BASAL TUBERCULOSIS

Tuberculosis of the lower lobes, without coincident involvement of the apices is a more frequent happening in children than in adults. Not only the primary lesion but also later infection of the lung may have such a localization. Some of these basal infiltrations may be explained as an extension of the inflammatory process from tuberculosis of the hilum into the lower lobes. In other cases undoubtedly the lobe becomes involved independently. The Roentgen

appearance in these cases is that of a consolidation of the lower lobe which presents no point of distinction from ordinary pneumonia. For this reason its tuberculous character cannot be determined at once. Here the evidence derived from the clinical symptoms and signs outweighs the Roentgen findings and we are usually compelled to observe the further course of the disease, especially in respect to the persistence or disappearance of the infiltration, before any conclusions as to its nature may be drawn.

MILIARY TUBERCULOSIS

Miliary tuberculosis of the lungs occurs more frequently in children than in adults and in most cases it is but a part of a general miliary tuberculosis. It is thus often found in association with tuberculous meningitis. (Fig. 122.) In general, the Roentgen appearance of

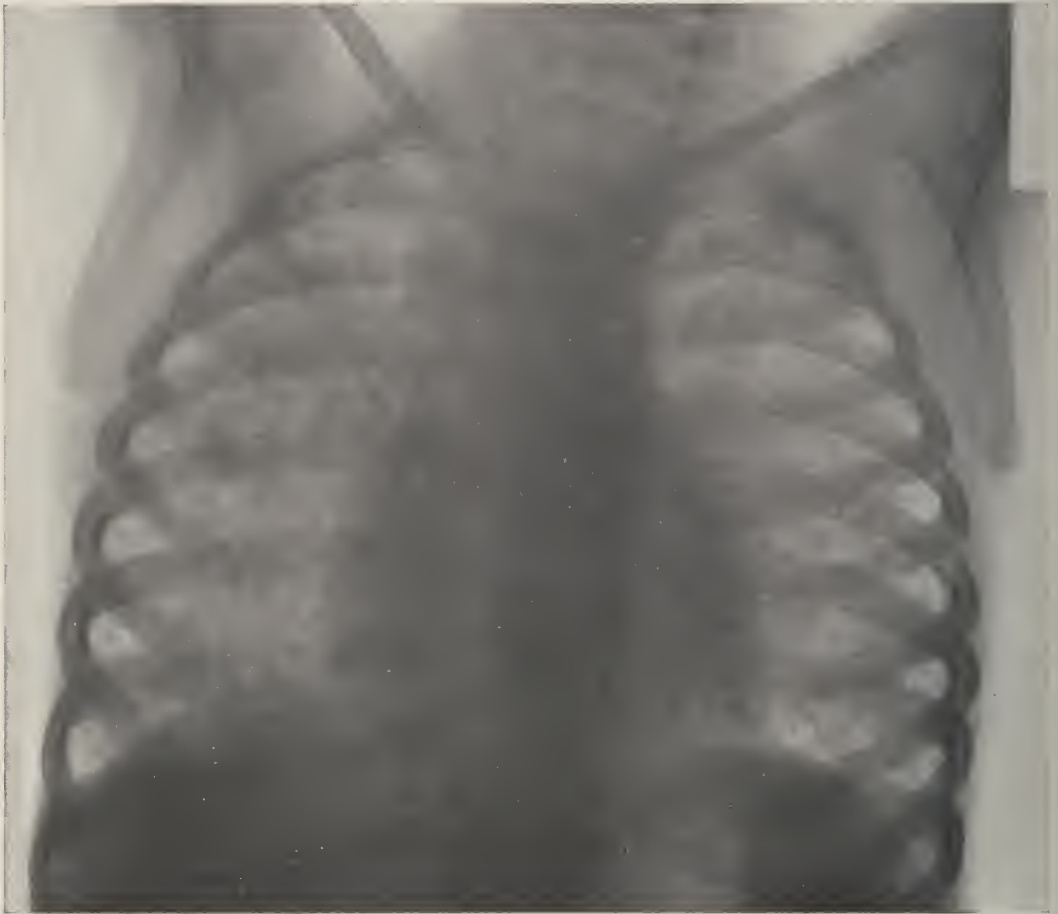


FIGURE 122
Miliary tuberculosis; conglomerate type.

tuberculosis in children is identical with that in adults. There is practically never evidence of an older lesion in the lungs, in spite of the fact that at autopsy small primary lesions are usually present.

In one respect the shadows of miliary tuberculosis in children may differ from those in the adult. In some cases, the tubercles are grouped into small masses surrounded by a zone of exudation so that conglomerate tubercles are formed. This is illustrated in figure 122, the plate of an infant who succumbed to the infection. At autopsy, the tubercles were not of the miliary type, but were larger and easily visible and agglomerated into masses by exudation about them. The softening of a subpleural tubercle may lead to a perforation of the lung and spontaneous pneumothorax. (Fig. 286.) The purely pulmonary form of miliary tuberculosis is very uncommon; as it is usually secondary to an older focus in the lung, it is more apt to develop in later childhood.



FIGURE 123
Miliary tuberculosis

The infiltrations are less regular in distribution and there may be some evidence of caseation or fibrosis. (Fig. 123.) These cases often date their first symptoms from such diseases as measles or whooping cough which, as is well known, predispose to a dissemination of the tuberculous infection. In this child, who was nine years of age, the pulmonary symptoms followed an attack of measles. In spite of the extensive involvement of the lungs the temperature range was not high and the only physical signs were slight dullness over the right upper lobe with a few subcrepitant rales.

ADULT FORMS OF TUBERCULOSIS IN CHILDREN

The types of tuberculosis which we have thus far described comprise its commoner forms, which are met with during infancy and childhood. As we approach puberty we encounter occasionally



FIGURE 124

Adult form of tuberculosis in child. Disseminated lobular infiltrations with beginning fibrosis in apices with cavity formation.

examples of the disease in which are seen evidences of greater chronicity and a tendency to induration which we have found so characteristic of adult or tertiary tuberculosis. For reasons which are still but imperfectly understood the apical regions are especially susceptible to tertiary tuberculosis and we there find evidence of its earliest development. (Fig. 124.) The subsequent course of apical tuberculosis in the child is similar to that in adults and for this reason an extended description of it is omitted. The antagonistic influences of caseation and fibrosis may here also result in the multiform pictures of fibro-caseous tuberculosis with cavitation, (fig. 125), and lobular or broncho-pneu-



FIGURE 125

Adult type of tuberculosis in a young child. Fibro-caseous tuberculosis with cavitation in upper lobe.

monia. (Figs. 126 and 124.) We must, however, regard these adult types of the infection as very exceptional and when they occur in early childhood, as analagous to the precocious tertiary manifestations of syphilis. We may appropriately conclude our subject by a recital of



FIGURE 126

Disseminated tuberculous lobular pneumonia. Adult type.

two cases, which illustrate the evolution of the tuberculous infection in children as it was observed over a period of years. The first child came under observation at the age of six, when she presented the usual symptoms of tuberculosis of the bronchial lymph nodes. The plate, (fig. 118), showed in both lower lobes the calcified remains of small initial lesions which had apparently run a symptomless course. There was in addition a marked enlargement of the bronchial nodes in which lime deposits were already visible. After a lapse of six years, during which she was in good health, she was reinfected. She became acutely ill and presented the signs of an apical process. On the plate, (fig. 127), we



FIGURE 127

Evolution of tuberculosis in adolescent child, showing development of an apical cavity. Previous plate, (fig. 118), made 6 years before shows only tuberculous adenopathy.

see a typical adult form of tuberculosis, consisting of nodular infiltrations in the right apex surrounding a circular cavity.

The second case illustrates in an even more complete manner the cycle of changes which ran their course in three years. We have already alluded to the symptoms which ushered in the disease in this child of eleven years as a primary lesion of the right lower lobe. (Fig. 105.) After the subsidence of the acute symptoms of the onset of the infection, this child remained apparently in good health for three years.

although her lungs bore the fruits of the disease in a marked adenopathy. Finally at the age of fourteen she once more presented herself with the signs of an apical infection. We now find on the plate the typical nodular infiltrations of adult tuberculosis at both apices, including a cavity at the left apex. (Fig. 128.)



FIGURE 128

Evolution of tuberculosis in a child. Previous plate, (fig. 105), showed initial lesion and glandular enlargement. In this plate, 3 years later, upper lobe infiltration with cavity has developed together with marked increase in size of nodes. Arrow points to large paratracheal nodes.

CHAPTER VII

Pneumonia

LOBAR PNEUMONIA. The Roentgen shadow of a lobar pneumonia owes its more or less characteristic appearance both to the nature of the pulmonary exudate and to its lobar distribution. To the former we can ascribe the homogeneity of the shadow which results from a consolidation of the lung; the latter accounts for its configuration which is determined by the shape of the individual lobe or lobes which are affected by the disease.

We must however not expect to find an exact correspondence in shape between the pneumonic shadow and the lobe of the lung, because a number of factors come into play which may considerably modify the former. In the first place, a lobe may be only partially consolidated so that its Roentgen shadow will not be coextensive with nor have the exact shape of the lobe. This is particularly true of upper lobe pneumonias in children in whom the apices are often not involved. Of greater importance in modifying the pneumonic shadow is the position of the diseased lobe relative to the adjacent uninvolved ones. Owing to the oblique plane of the main interlobar fissure, the lobes overlap



FIGURE 129
Pneumonia of right upper lobe

each other at certain levels. Wherever this overlapping occurs, the shadow cast by a consolidation of an individual lobe is attenuated and its contour altered by the superjacent air-containing lung. It will also be apparent that the height of the target of the tube with respect to the consolidated area and the position of the patient, whether this be dorso-ventral or the reverse, will exert a marked influence on the size and appearance of the pneumonic shadow.

UPPER LOBE PNEUMONIA. A consolidation of the right upper lobe usually casts a well defined Roentgen shadow in which the lower limit of the lobe is clearly outlined. (Fig. 129.) This is not the case on the left side because here the interlobar fissure is oblique so that the lower border of the pneumonic shadow becomes somewhat hazy. It will be noted that in many upper lobe pneumonias the apex remains uninvolved to the end of the disease. This is particularly the case in infants and young children in whom the consolidation may not be truly



FIGURE 130

Pneumonic triangular infiltration in right upper lobe in a young child.

lobar but rather has a triangular shape with its base to the axilla. (Fig. 130.) Later in the progress of the disease the triangle may encroach on the apical region without however completely filling it. (Fig. 131.)

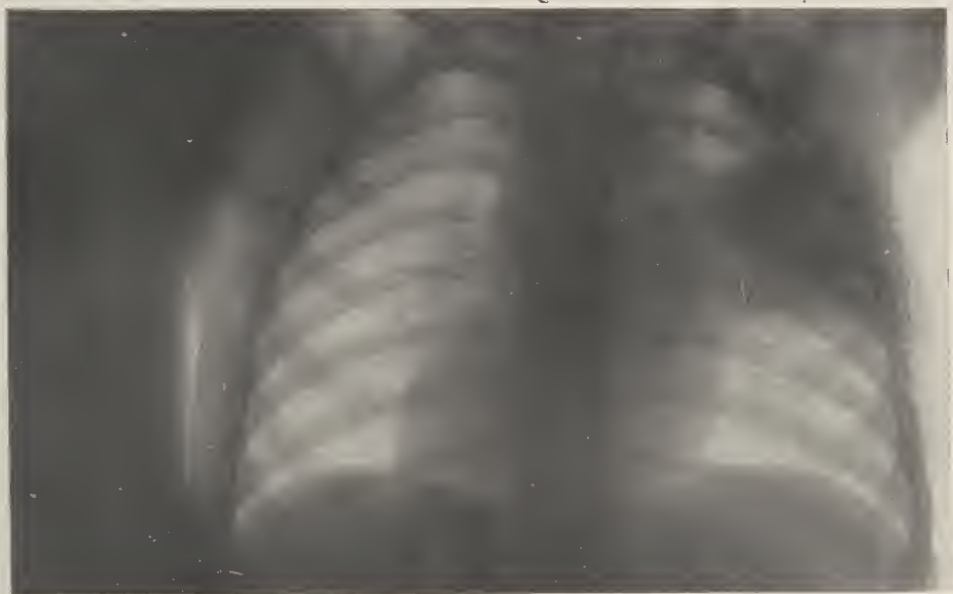


FIGURE 131

Partial consolidation of right upper lobe. Ventro-dorsal view.

An isolated pneumonia of the middle lobe is extremely rare. Its shadow, in the dorso-ventral direction, which is best adapted to bring it out, is characteristic. (Figs. 158 and 132.) It is not imabobable that it occurs more often in association with disease of the lower lobe in which case it merges with the shadow of the lower lobe pneumonia and is correspondingly modified.



FIGURE 132

Pneumonia of the middle lobe.

LOWER LOBE PNEUMONIA. The most varied shadows are found with pneumonia of the lower lobes, depending on the extent of the consolidation, the stage of the disease and on the technique of the exposure. Thus in figure 133 is seen a typical sharply outlined consolidation of the right lower lobe, the tube being focussed relatively



FIGURE 133

Right lower lobe pneumonia; high focus of tube.

high near the level of the interlobar fissure posteriorly. On the other hand, in figure 134, the tube is in a lower position so that the margin of the pneumonic shadow is projected upward and attenuated by the

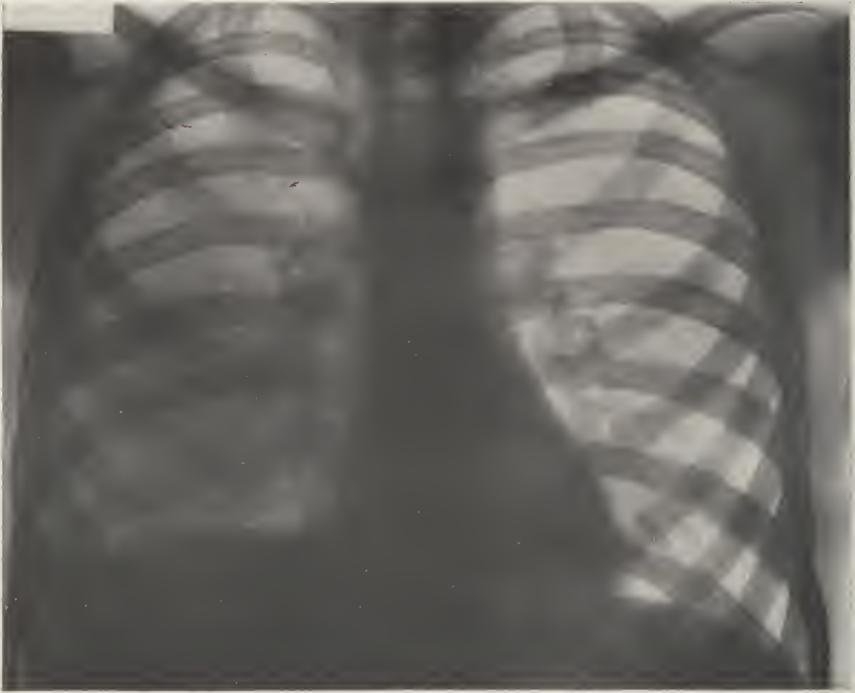


FIGURE 134

Lower lobe pneumonia at an early stage of disease. The shadow is extensive and its upper portion is indistinct due to the low focus of tube. Interlobar fissure outlined.

interposed, aerated upper lobe. When the tube is focussed on the cervical region the shadow may be projected so far downward that it may be entirely obscured by the diaphragm in expiration or it may appear as only a narrow band above the latter and parallel with it. In the ventro-dorsal position, owing to the closer proximity of the lower lobe to the plate, the pneumonic shadow is of greater extent. With a low tube target the shadow may be projected almost to the apex as though there were a consolidation of the entire lung. Under these circumstances, however, the upper border of the shadow lacks the sharp definition which is obtained with a higher position of the tube. (Fig. 135.)



FIGURE 135

Pneumonia of left lower lobe. Owing to the low tube position, the pneumonic shadow is projected to the level of the second rib anteriorly.

In numerous instances the shadow of a lower lobe pneumonia does not present a completely lobar form. The lung may be only partially consolidated as in figure 136, in which the periphery is uninvolved. Occasionally a triangular shadow similar to that in the upper lobes may be found in the axillary portion of the chest just above the diaphragm.



FIGURE 136

Right lower lobe pneumonia with partial consolidation.

Multiple lobe involvement, either simultaneous or in succession is often seen. Thus in figures 137 and 138 there is a consolidation of both the upper and lower lobes on the right side, whose shadows can easily be distinguished.

The shadow of a lobar pneumonia is usually homogeneous. In its earliest stages it is of moderate density so that the lung structure may be distinctly seen through it. (Fig. 134.) As the disease progresses toward gray hepatization, the shadow increases in density. With rare exceptions it is not as dense as the liver, whose dome can always be distinguished from the consolidated lung; the ribs are also visible through it. Shadows in the upper lobe are of greater intensity than those in the lower lobes.

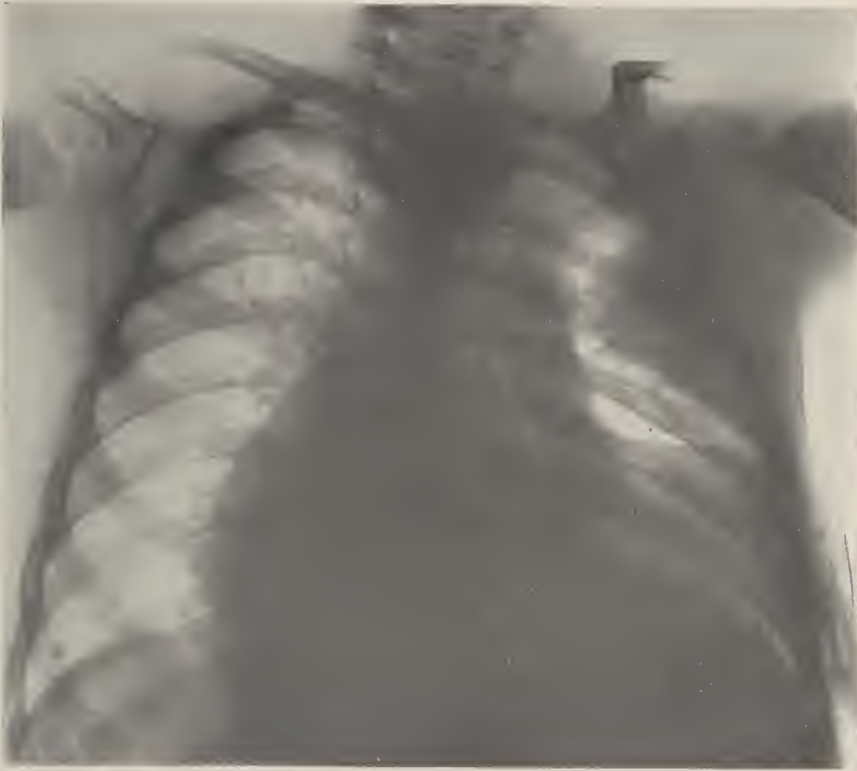


FIGURE 137

Right upper and lower lobe pneumonia. Ventro-dorsal view.



FIGURE 138

Pneumonia of right upper and lower lobes. Middle lobe not involved.

The resolution of a lobar pneumonia, which can be observed by successive Roentgen examinations, presents numerous points of interest. As a rule, the absorption of the exudate begins at the periphery of the lung and progresses toward the root (Fig. 139) ; it may, however, begin



FIGURE 139
Resolution of lower lobe pneumonia.

in any portion of the diseased lung. Thus in figure 148 there is an atypical absorption of the exudate, which began at the base and gradually extended upward. Complete resolution may require several weeks and distinct shadows are almost invariably found on the plate long after all physical signs of the disease have disappeared. In fact it is not uncommon for the affected lung to retain a mottled appearance due to deficient aeration for a considerable period after a pneumonia. Such

a mottling, unless the examiner is informed of the existence of a recent pneumonia, may give rise to errors in interpretation. Thus in figure 140 the left upper lobe exhibits a fine granulation which is not unlike

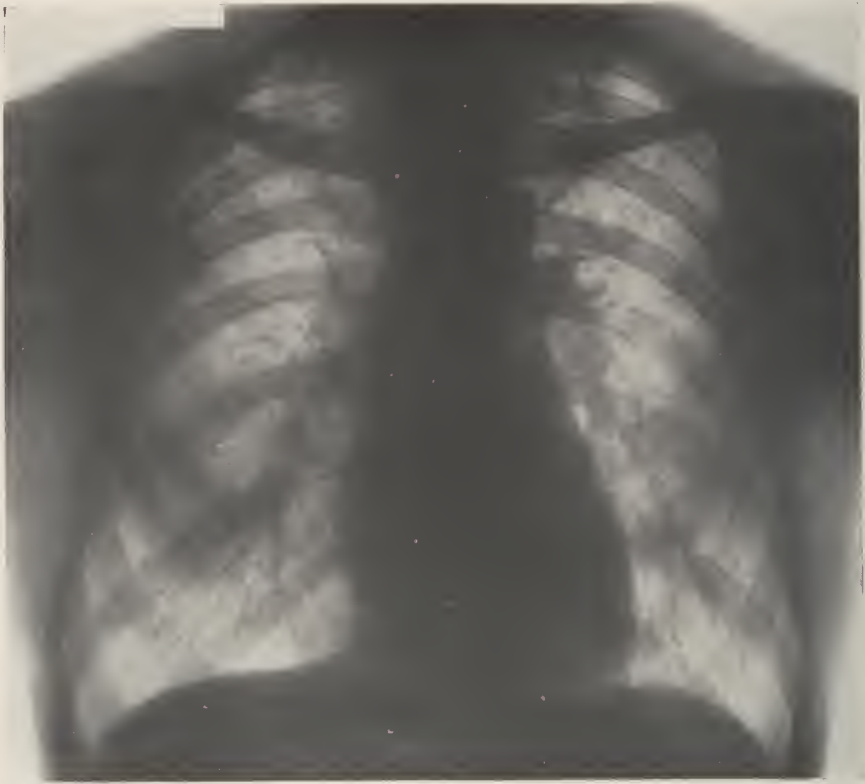


FIGURE 140

Mottling of left upper lobe after pneumonia, closely resembling a miliary tuberculosis.

that of a miliary tuberculosis. It persisted for a few weeks after a lobar pneumonia and finally disappeared entirely.

At times the progress of resolution is very irregular; it may be much more rapid in some portions of the lungs than in others, so that a small area may have entirely cleared up while the surrounding lung is still consolidated. There may thus be formed circular defects or pseudocavities which it is important not to confuse with abscess cavities. (Fig. 141.) In general, the shadows disappear most rapidly from the



FIGURE 141

Atypical resolution of a lobar pneumonia. The light area within the consolidated lung simulates an abscess cavity.

periphery of the lung; as the hilum is approached resolution is retarded and there will occasionally be noted a persistence of the shadows at the root for a number of weeks. (Fig. 142.) If the patient is observed for the first time at this stage of the disease, the Roentgen picture may be



FIGURE 142

Residual pneumonia at the root of the right lung

very confusing. For example, in figure 143, there is a homogeneous, sharply outlined shadow extending outward from the root of the right lung, which by itself might pass for a mediastinal tumor. Resolution of



FIGURE 143

Slow resolution of a pneumonia with a persistence of the infiltration in the mesial portion. Note unusual sharp delimitation of pneumonic from normal lung. See figure 144.

the pneumonia was arrested at this stage for over a week, when it was rapidly resumed. A second plate, made two weeks later showed a small, residual hilum pneumonia. (Fig. 144.) During resolution circu-

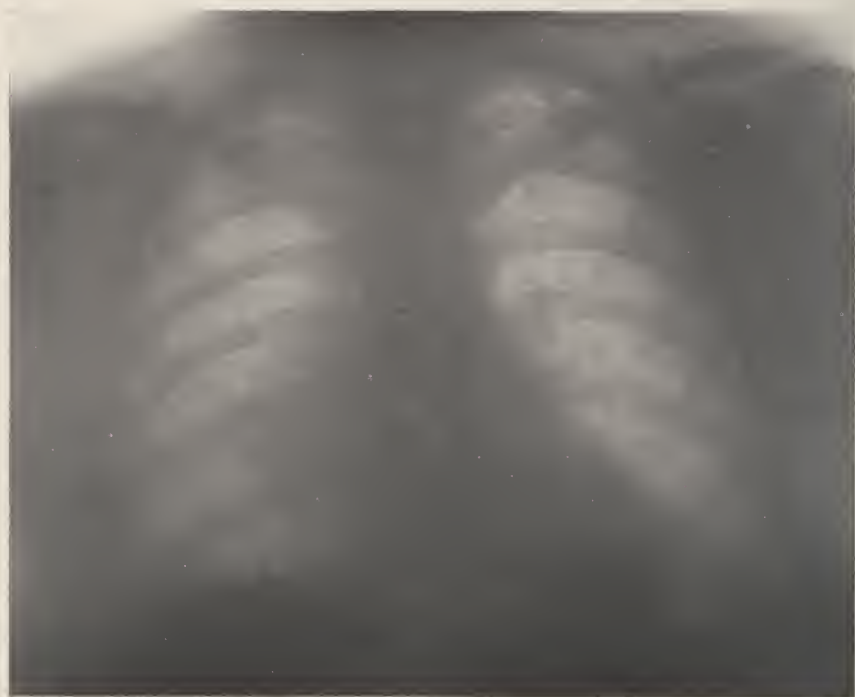


FIGURE 144

Same case as figure 143 two weeks later, showing further resolution. Small residual infiltration at right hilum.

lar islets of infiltration may be left within the lung, which may so closely resemble new growths that only their subsequent disappearance will clear up their true nature. The semi-circular consolidation seen at the right border of the heart in figure 145 persisted for weeks after a pneu-



FIGURE 145

Residual pneumonic area along right border of heart suggesting a tumor. The shadow disappeared one week later.

monia and was the occasion for the liveliest speculation until it finally disappeared. Occasionally bizarre shadows of resolving pneumonia may be observed as in figure 146.

Associated with pneumonia of the lower lobes, there is commonly an immobility and elevation of the diaphragm on the affected side which is presumably dependent on a dry pleurisy. This immobility is present not only during the active stage of the disease but may persist for some weeks after complete resolution. We may thus be enabled from the Roentgen plate alone to infer the previous existence of a pneumonia in cases which have undergone complete resolution, or in which a few shadows of doubtful significance are seen at the base. The

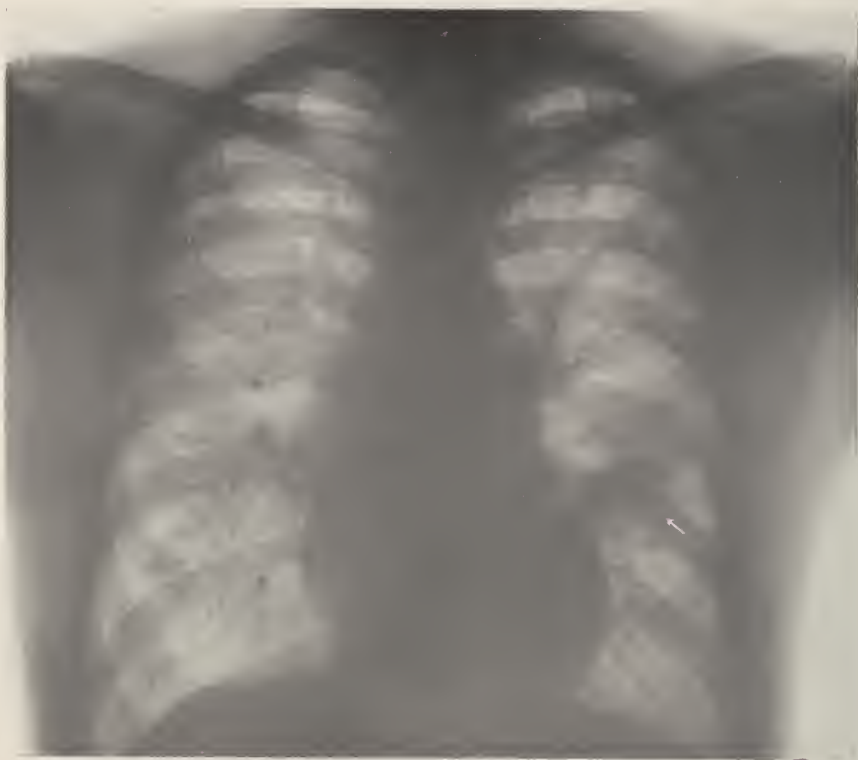


FIGURE 146

Unique shadow of residual pneumonia along left border of the heart.

pleural complications of pneumonia, especially empyema, will receive detailed consideration in a separate chapter. Reference may here be made to those cases of pneumonia in which the pleura is covered by such a thick exudate that the physical signs of an effusion are simulated. On the plate also the differentiation may be difficult or impossible. The shadow of a pleuro-pneumonia is as dense as that of an effusion and for this reason the diaphragm may be invisible through it. In the case illustrated in figure 147 the massive shadow in the right chest was presumed to be due to fluid, a presumption which received much support from the physical examination. Yet, repeated aspiration was fruitless



FIGURE 147

Pleuro-pneumonia. The dense shadow was due to a very thick pleural exudate. Repeated aspiration was negative. See figure 148.

and the subsequent resorption of the exudate, beginning at its most dependent point, figure 148, clearly proved that the shadow was not due to fluid in the pleural cavity.



FIGURE 148

Pleuro-pneumonia showing unusual resolution beginning at the base and extending upward. Same case as figure 147, two weeks later.

THE RELATION OF THE ROENTGEN TO THE PHYSICAL DIAGNOSIS OF PNEUMONIA

In the case of adults, the opportunity is rarely given to examine with the Roentgen Ray the earliest stages of lobar pneumonia. For this reason the relation of the development of definite consolidations to the physical signs is a matter difficult of determination. It seems to be true, however, that lobar shadows on the plate are at least contemporary with the physical signs of pneumonia and it is likely that in many cases, the former precede the latter. The discovery of a lobar pneumonia on the screen or plate one or more days before the appearance of physical signs, has given a renewed interest to the conception of pneumonia as

originally a central process which only later reaches the surface of the lung, a conception which in recent times has fallen somewhat into disfavor among clinicians. For we occasionally see cases of pneumonia, showing extensive shadows on the plate, located at the root of the lung, which are difficult to explain otherwise than as deeply seated or central pneumonias. Thus, in figure 149 the right hilum pneumonia, which at



FIGURE 149

Hilum or central pneumonia, probably one day old. Process later extended to periphery.

the time of the examination was perhaps one day old, produced negligible physical signs. Only after its gradual extension to the surface and with an involvement of the peripheral portion of the right lower lobe did outspoken signs of consolidation appear.

In infants it is well known that Roentgen shadows frequently precede the physical signs of pneumonia. The localization of the pneumococcic infection in the lung may remain clinically latent and may not be discovered by physical examination for days after a shadow can be distinctly seen on the fluoroscopic screen. In older children especially,

this fact has an important practical bearing. For example, one of the difficult problems which is not infrequently encountered both by physicians and surgeons, is the differentiation in children between lower lobe pneumonias on the one hand and intra-abdominal conditions, such as peritonitis, especially of the pneumococcus type and appendicitis, on the other. The well known tendency of the pleuritic pain of a lower lobe pneumonia to be referred to the abdomen and to produce the reflex signs commonly associated with peritoneal irritation, may make it impossible clinically to distinguish between the two. This is the more true because the uncertainty arises usually during the earlier stages of pneumonia when physical signs may be entirely absent. In these cases the Roentgen examination is of incalculable service, as the discovery on the screen or the plate of a pneumonic consolidation may spare the child an unnecessary laparotomy. On the other hand, a negative Roentgen finding will confirm the diagnosis of intra-abdominal disease and lead to a timely operation. The Roentgen examination is peculiarly reliable in these cases because, in the earliest stages of lobar pneumonia encountered clinically, even before physical signs are present, well marked shadows are already to be seen.



FIGURE 150

Confluent lobular pneumonia, probably due to aspiration. Duration two months. See figure 151.

BRONCHO-PNEUMONIA AND LOBULAR PNEUMONIA

The shadows of a broncho-pneumonia are often not demonstrable by a Roentgen examination. Unless the areas of pneumonia are large or confluent, they will cast only indefinite shadows, which it is difficult to distinguish from the normal lung markings. This is particularly true in children. The common location of broncho-pneumonia at the bases of the lungs, adds to the difficulties in diagnosis, because most of the lesions are apt to be deeply situated and therefore will be obscured by adjacent areas of normal or emphysematous lung. It is a common experience therefore, for the clinician to obtain physical signs of a bronchiolitis and lobular pneumonia when the Roentgen examination is entirely negative.

In cases of confluent lobular or broncho-pneumonia on the other hand, the shadows may be very dense and extensive, involving commonly the basal portions of the lungs. In figure 150 is seen such an extensive broncho-pneumonia of both lower lobes which followed prolonged submersion in water. The clinical course was prolonged for over two months and during it there was much cough and dyspnoea. The gradual resolution of the process is indicated in a subsequent plate. (Fig. 151.)



FIGURE 151

Same case as figure 150 showing slow resolution.

In so-called ether or postoperative pneumonias similar shadows, though of less intensity, are frequently found in one of the lower lobes.

The introduction into the community during the past few years, of epidemic influenza with its accompanying pneumonia has necessitated a change in our ideas with regard to the Roentgen diagnosis of lobular and broncho-pneumonia. The pneumonias which complicate influenza are characterized by an exudate which has great absorptive powers for the Roentgen ray so that shadows of great density are produced. Further, the diversity of the pathological processes, comprising



FIGURE 152

Confluent lobular pneumonia of right upper and lower lobes in epidemic influenza. Appearance indistinguishable from tuberculosis.

hemorrhagic, exudative and purulent elements, scattered indiscriminately throughout the lungs, has been responsible for a multitude of Roentgen types which were previously not encountered. Lobular areas may coalesce to form pseudo-lobar consolidations which cannot be distinguished from true lobar pneumonias. This process frequently affects more than one lobe as in figure 152 and there is also a common involvement of the upper lobes. In some forms of the disease, the individual infiltrations remain small and discrete and their general appearance and frequent occurrence in the upper lobe creates the greatest difficulty in distinguishing between influenza pneumonia and tuberculosis. The presence of hemoptysis may add to this difficulty so that during epidemics the decision between tuberculosis and influenza has



FIGURE 153

Persisting universal broncho-pneumonia. Cardiac enlargement.

to be left open pending further Roentgen examinations. In the case illustrated in figure 152 both the clinical picture and the Roentgen plate were compatible with a tuberculous process in both upper lobes. Yet, the occurrence of the illness during an influenza epidemic made it advisable to await the outcome, before definitely affirming its tuberculous nature. The wisdom of this course was confirmed by the favorable termination of the disease within a few days when a second Roentgen examination showed a complete disappearance of the pneumonic shadows.

Although the rapid disappearance of the shadows of influenza pneumonia may thus be regarded as a proof of their non-tuberculous character, we must bear in mind that in occasional cases, there is an imperfect resorption of the exudate together with interstitial fibrous changes, which may make for a persistence of the Roentgen shadows for a shorter or longer time. Thus in figure 153 there is an almost universal lobular pneumonia which was still present four weeks after the subsidence of the fever. The patient suffered from marked dyspnoea which could readily be explained by the extent of the pulmonary involvement and perhaps also because of obliterating bronchiolitis. The constant, extreme dyspnoea lasting for weeks, had apparently effected a weakening and dilatation of the right heart.

One of the surprising disclosures of the Roentgen examination is the frequency with which unsuspected broncho-pneumonia at the bases is found in patients who are clinically regarded merely as cases of acute or chronic bronchitis. These pneumonias are commonly so mild and their physical signs are so completely obscured by those of bronchitis and emphysema, that they are overlooked and the symptoms are therefore regarded as an exacerbation of an existing bronchitis. The shadows of the pneumonia, usually in one of the lower lobes is of course readily perceived on the plate. Frequently the persistence of these pneumonic infiltrations is responsible for a chronic cough with expectoration which, in the absence of physical signs, is apt to put the patient under suspicion of tuberculosis; the Roentgenologist will here frequently have the grateful task of dispelling the fear of tuberculosis and of giving the proper direction to the treatment.

CHAPTER VIII

Pulmonary Suppuration—Abscess and Gangrene of the Lung

Suppurative lung disease includes a number of affections of diverse etiology whose common basis is an inflammatory process which leads to necrosis of the lung parenchyma. Clinically we further differentiate them into cases of simple abscess and of gangrene of the lung. The latter are dependent on invasion of the lung by the micro-organisms of putrefaction which impart to the sputum its fetid odor, on which the diagnosis of gangrene is essentially based. Here also, as in the diagnosis of tuberculosis, it is necessary to be informed in regard to the pathology and clinical aspects of the disease, if the Roentgen shadows are to be intelligently interpreted. There will be frequent occasions when the plate will exhibit shadows which are not characteristic of lung abscess and the distinction between it and other disease will not be obvious. The Roentgenologist will then have to draw on his knowledge of the pathology of the disease and of the clinical symptoms in the particular case in order to eke out the diagnosis.

For practical purposes we may classify cases of lung suppuration roughly into two groups, the larger one of which includes the abscesses which develop acutely and are characterized by an early onset of gangrene and the formation of one or more necrotic cavities. There is reason to believe that the cause of the suppurative process in these patients is the aspiration of septic material from the mouth, especially during anesthesia or other unconscious states, with the growth in the bronchi and lungs of anaerobic bacteria.

In contrast to this group stands a second in which the disease is slower in its development, whose pathogenesis is probably quite different. The most prominent members of this group are the cases of pneumonia which have become chronic and have later, as an incident to fibrosis and bronchial dilatations, developed a secondary gangrene and abscess of the lung.

It will contribute to a clearer understanding of the manifold causes of lung abscess and its probable mechanism if we indicate, by a tabulation of the first 100 cases which we observed, the various causative factors which preceded their onset.

Aspiration Abscess—

Post Operative:

Tonsillectomy	21
Other operations	6

Following Unconscious States:

Submersion	2
Morphine	1
Alcoholic	1
Foreign Body	2
Acute Pneumonic Gangrene	16

Non-Aspiration Abscess—

Chronic Pneumonic	21
So-called Grippe	21
Empyemic	1

Miscellaneous—

Embolic (Suppurative Thrombo-phlebitis)	1
Tuberculous	2
Secondary to Carcinoma of Lung	1
Secondary to Carcinoma of Esophagus	1
Actinomycotic	1
Diabetic	1
Syphilitic	1

As will be seen in the development of our theme, this division into aspiration and non-aspiration abscesses, in which the majority are comprised, not only bears on their causation and mechanism but is also reflected in the pathology of the disease, its mode of progression in the lung and therefore also in its Roentgen appearance.

PATHOLOGY

Let us rehearse briefly the probable sequence of events which terminate in an acute lung abscess. If we conceive this as due to the aspiration of infectious material, which, in our experience is most often derived from diseased tonsils, during operation, we may picture to ourselves the lodgment of this material in one of the smaller bronchi. At the point of its arrest there develops an acute inflammation of the bronchial wall and a pneumonia of the adjacent lung. Of great significance for the further course of the disease is the presence in the aspirated material of putrefactive bacteria, which exert their destructive influence on the wall of the bronchus, which they weaken and dilate and finally destroy. The termination of this process, which is commonly a rapid one, is marked by the formation of a smaller or larger cavity, whose walls are formed by the consolidated and necrotic tissue

in which it has been excavated. This consolidation, consisting of a pneumonic infiltration may involve a small portion of a lobe or a whole lung. The further course of such an abscess may result either in complete resolution or in early death or finally in a subacute and chronic stage.

It is important to remember, and this has a bearing on the Roentgen interpretation, that the pneumonic process which is invariably associated with a lung abscess, exercises a determining influence on the further course of the case. This pneumonic process, under the stimulus of the gangrenous focus in its centre, may acquire a persistent, chronic character which leads to induration and occasionally, in long standing cases, becomes fibrotic. Conditions are therefore favorable for the secondary formation of multiple, cylindrical dilatations of the bronchi which may be infected by anaerobic organisms and may in turn be converted into necrotic cavities. We therefore usually find in these cases one large cavity which is the original one and numerous smaller gangrenous bronchiectases.

In the non-aspiration cases, the course of the disease is frequently more chronic from its inception. Primarily the process is not suppurative or gangrenous. There is an insidious development of a subacute or chronic pneumonitis with fibrosis, which, as in the other forms of the disease, is responsible for multiple bronchial dilatations. The disease may be arrested at this stage for a variable period of time, for months or years, during which evidence of suppuration or gangrene of the lung may be absent. It is only with the advent of putrefactive bacteria, under conditions which we cannot explain that the disease assumes a really suppurative and destructive form; it then becomes clinically indistinguishable from cases of aspiration abscess.

It will be evident from this short description of the pathology of lung suppuration, that putrefactive bacteria play a dominating role in the evolution of the disease. In fact, the essential element in acute or aspiration abscess is putrefaction, which is rarely or never absent. We may therefore properly speak of it as an acute gangrenous pneumonitis, which in our experience has a definite incubation period and a unique pathology. The external evidence of the gangrene, consisting in a fetid odor to the breath and sputum, appears regularly on the 12th to the 14th day from the time of aspiration, be this postoperative or from some other cause.

ROENTGEN DIAGNOSIS

Let us now inquire in what manner a lung abscess reveals itself on the Roentgen plate. Its earliest stage can best be observed in the case of acute or aspiration abscess, as the chronic types do not come under early observation. If patients are examined even a few weeks after

the onset, there will usually be found a cavity of varying size, surrounded by an area of pneumonia. There is no constant relation between the size of the cavity and that of the surrounding pneumonia. Frequently they seem to stand in inverse relation to each other. Thus the cavity in figure 154 which is of moderate size, is surrounded by a

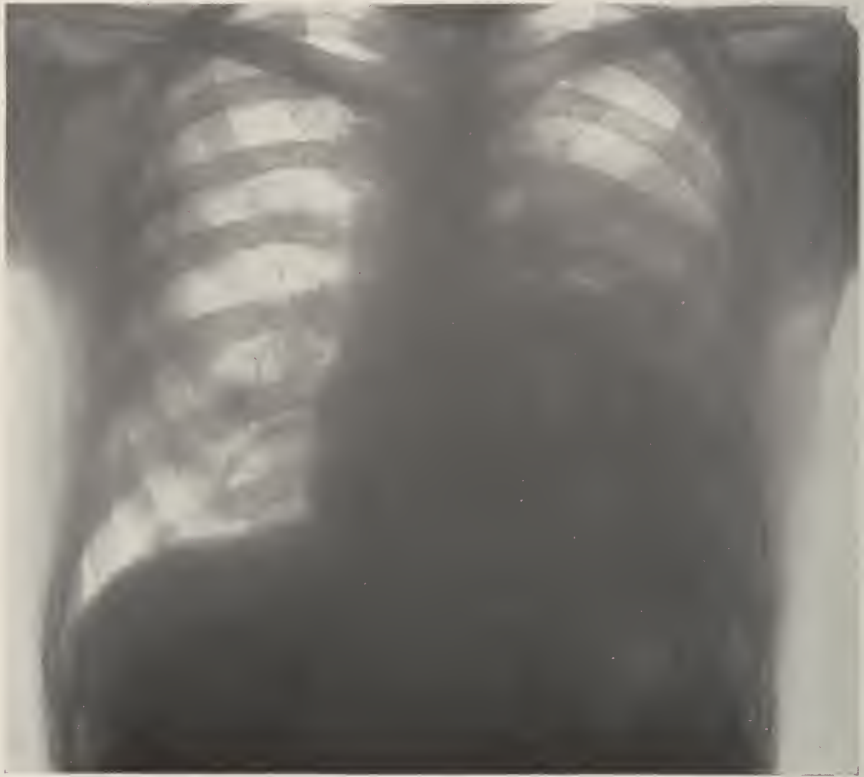


FIGURE 154

Abscess cavity surrounded by an extensive area of consolidation. Duration eleven weeks.

consolidation of the entire left lower lobe. In this case the disease was of eleven weeks' duration. The excavation of the lung may reach great proportions very early in the disease. The abscess cavity in figure 155 which is half full of fluid must have resulted from a rapid breaking down of the lung, as it had already attained this size three weeks after



FIGURE 155

Lung abscess three weeks after tonsilectomy.

tonsillectomy. Again in figure 156, six weeks after the onset, we have a complete destruction of the right upper lobe which is converted into a cavity half full of fetid secretion. It is likely that these large cavities are formed by the rapid necrosis of an extensive pneumonic area which must have preceded them.

The typical finding therefore in the early stages of acute abscess and gangrene of the lung, in those cases which are not progressing to cure, is a large cavity usually partly filled with fluid, surrounded by more or less pneumonia. These patients are usually acutely ill, have high fever and expectorate large quantities of characteristically foul sputum and they not uncommonly succumb at this time from a putrid intoxication.

When we turn now to the chronic postpneumonic type of abscess, we are struck by the preponderance of pneumonic infiltration and by the minor role which the necrotic process may play. For a long time the plate may disclose no evidence of suppuration or cavity formation, but only a pneumonic infiltration at the base. In fact, during this insidious

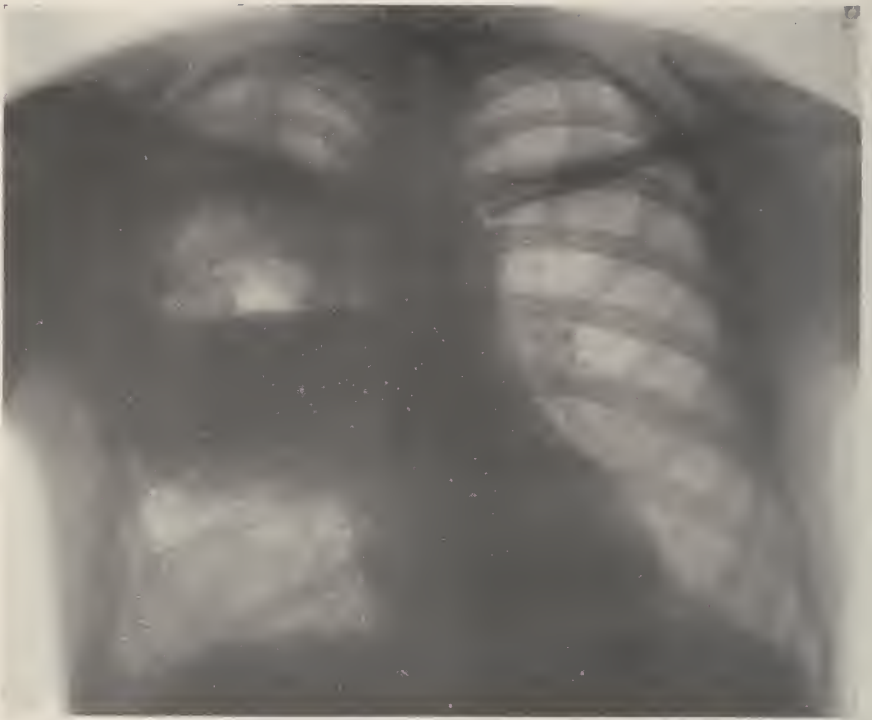


FIGURE 156

Excavation of the entire right upper lobe by an acute abscess in six weeks.

development of a chronic pneumonia, it is only potentially a case of lung suppuration; the conditions which will favor a secondary anaerobic infection are in process of creation. It is even probable that the indurated lung offers a greater resistance to necrosis than an acutely inflamed one, so that the cavities which are formed are small and slow in their evolution. The small irregular gangrenous bronchiectases are frequently entirely obscured by the surrounding lung. For this reason, the clinician will often detect the signs of suppuration before typical evidence of it is found on the plate.

The development of fibrosis in the pneumonic lung may be traced by successive Roentgen examinations until dense irregular shadows, such as are seen in figure 157, are found. We have in this case a lung gangrene which terminated a persistent pneumonia of eight months' duration. Although no cavities were visible on the plate, the profuse purulent and fetid expectoration left no doubt of their presence and this was confirmed by the necropsy findings.

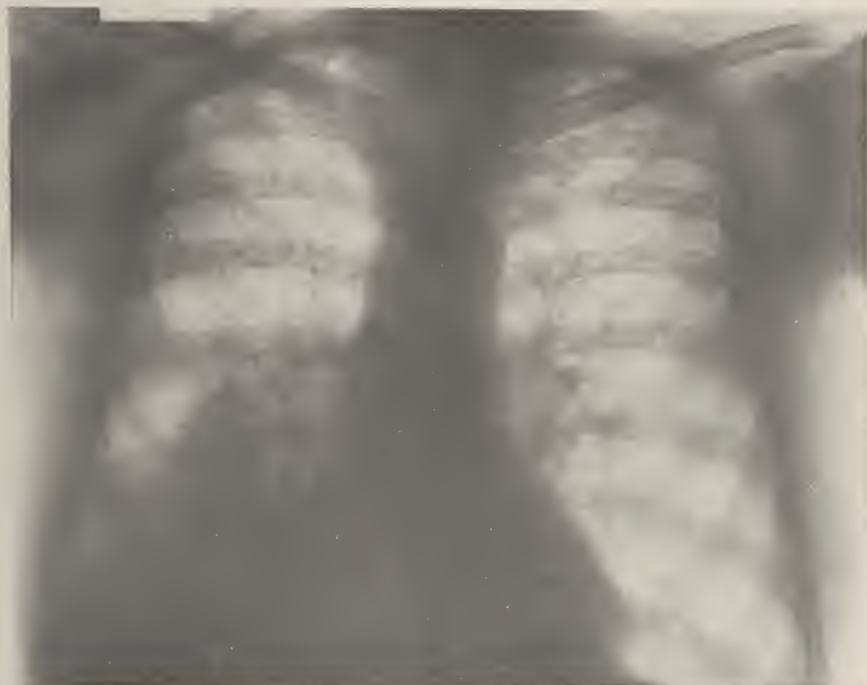


FIGURE 157

Indurative pneumonia with secondary gangrenous bronchiectases. Eight months' duration.

While the suppuration of chronic pneumonias involves most often the lower lobes, this situation of the process has numerous exceptions. The upper lobes may be the seat of the disease and in rare cases, there may even be an isolated involvement of the middle lobe. Because of its great rarity we illustrate such a case in which the Roentgen plate shows the characteristic appearance of a middle lobe pneumonia. (Fig. 158.) In this patient the disease, of insidious onset, progressed gradually to the complete picture of lung suppuration. Aspiration near the right border of the sternum in the 4th interspace revealed a collection of pus in this region which at operation was found to be a subpleural abscess cavity of the middle lobe. Here again the outlines of the cavity were obscured by the shadow of the infiltrated lung.



FIGURE 158

Indurative pneumonia of middle lobe with abscess.

THE FURTHER COURSE OF ASPIRATION ABSCESS

Acute lung abscess may result fatally within a short time during the stage of extensive necrosis and cavitation. When, on the other hand, the patients survive for months or years, as they frequently do, the inflammatory process undergoes an evolution which is reflected in a constantly changing clinical picture. In the portrayal of these changes the Roentgen examination can render us invaluable service. It faithfully depicts the extension or recession of the diseased area, it notes the existence and growth of cavities in situations inaccessible to physical examination and provides the only certain information as to the cure of the patient. Finally, by an accurate localization of the disease, it guides the surgeon in the precise application of his operative procedures.

We will attempt to reconstruct in a fragmentary way the life history of pulmonary abscess by the study of a number of plates which illustrate its various phases. The great role which the abscess cavities

and their highly infectious secretions play in the aggravation of the disease is constantly brought to our attention. The overflow of this secretion is an ever-present menace to adjacent or distant portions of the lung which are thus involved in the suppurative process. Figure 159



FIGURE 159

Abscess of left upper lobe with secondary infection and abscess formation of the lower lobe.

illustrates this clearly. The patient acquired an abscess of his left upper lobe following an operation. The contents of this abscess, whose fluid level is seen at the 2nd rib anteriorly, have infected the left lower lobe which is consolidated. A second cavity at the left base resulting from the necrosis of the pneumonic lung has already formed. Again in figure 160 we see a large cavity in an upper lobe which was presumably due to the aspiration of gastric contents incident to a spasmodic stricture of the oesophagus. For months this woman presented the symptoms of lung suppuration without, however, being acutely ill or

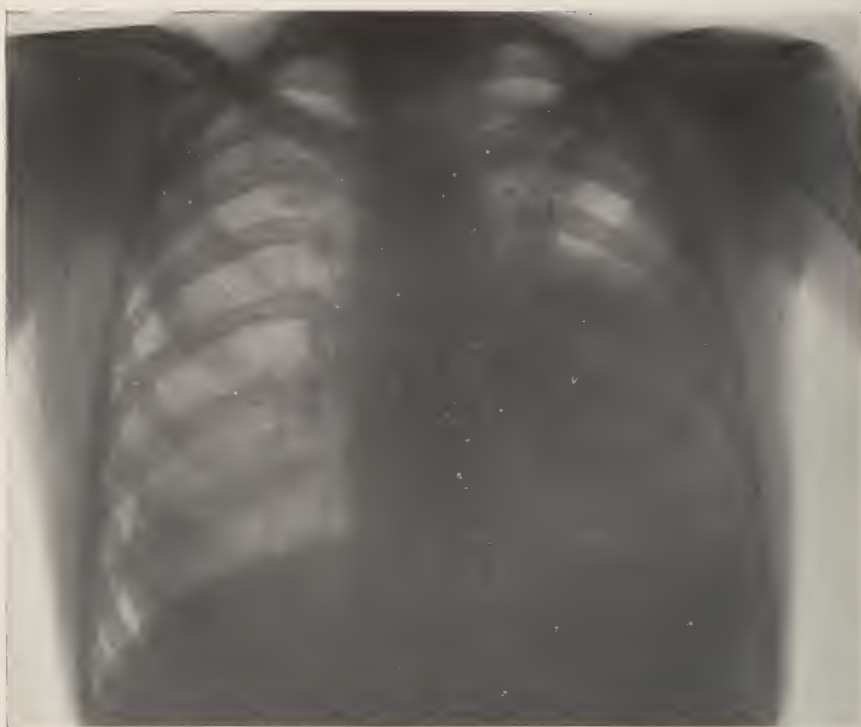


FIGURE 160

Abscess of left upper lobe. Secondary gangrenous pneumonia of lower lobe.

suffering from the effects of toxic absorption. There came a day however, when the purulent contents of the cavity infected the rest of the left lung. She became acutely ill, with signs of consolidation of her left lower lobe and died in a few days with the symptoms of putrid intoxication.

The clinical course of the disease is thus frequently punctuated by attacks of broncho-pneumonia which may undergo resolution or become necrotic; the exacerbation of cough, fever and expectoration may often be explained by such an extension of the process which is reflected on the plate by an increase in the size of the pneumonic area.

When acute abscesses progress to the subacute stage, it is usual for the infiltration to undergo considerable resorption so that the original cavity is surrounded by a relatively small area of pneumonia.

In fact, as seen in figure 161, the infiltration may entirely disappear and leave a walled-off cavity within the lung. It is probable that such a condition, unless the cavity itself becomes obliterated, is only temporary; the basis for infection of the lung still exists in the secretions of the cavity, whose pernicious effects sooner or later manifest themselves.



FIGURE 161

Chronic abscess cavity with well-marked capsule.

With increasing chronicity of the abscess, the fibrotic tendency of the infiltration becomes marked and secondary bronchiectases are formed. In the older cases therefore, multiple smaller cavities are usually found in addition to one large one. The lung abscess seen in figure 162 followed tonsillectomy and was of eight months' duration. The pneumonic area is well defined and encloses the original large cavity and two smaller secondary cavities above it.



FIGURE 162

Multiple abscess cavities in right upper lobe, eight months after tonsillectomy.

The extreme coarse fibrosis which is so characteristic of the fibroid form of tuberculosis, is rarely seen in chronic lung abscess. For this reason, marked deformity of the chest and displacement of the mediastinum are not often encountered. When this is the case, as sometimes happens, the distinction between suppuration and tuberculosis is difficult and final dependence must be placed on the clinical features. In figure 163 there is an extensive unilateral fibroid disease of the lung with multiple small cavities in the upper lobe. It will be noted that the right chest is smaller in volume than the left, the ribs are drawn together and the mediastinum is displaced to the right side. There is a noticeable absence of any disease in the left lung. The presumption of a long duration of the disease in this case is confirmed by the clinical history which informs us that the patient contracted her illness seven years before. During the interval she constantly expectorated purulent sputum which was occasionally fetid, in varying amounts up to ten ounces daily. We have here a form of chronic pulmonary suppuration which, because of its benign course, frequent hemoptysis and clubbed

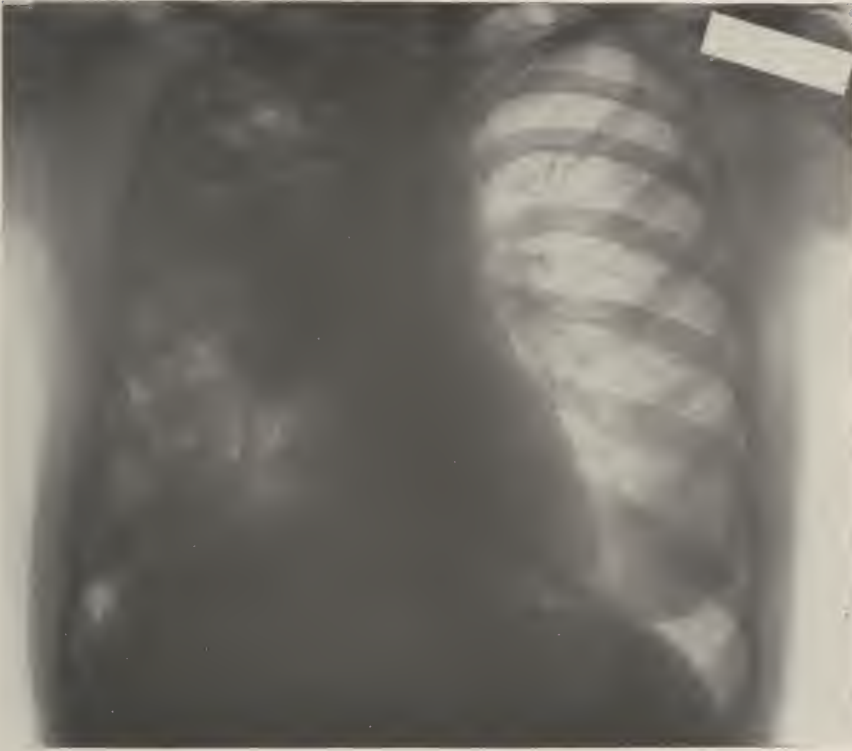


FIGURE 163

Chronic indurative pneumonia and fibrosis, with multiple bronchiectatic abscesses. Seven years' duration.

fingers is often treated as a tuberculous affection. When we weigh the conflicting evidence in a case of this sort, the absence of tubercle bacilli must not be permitted to bear too heavily in the scales; the possibility of secondary suppuration and gangrene engrafted on a tuberculous process has to be borne in mind.

While the distinction between tuberculosis and suppuration may therefore not at once be apparent from the plate, there are other cases, and these constitute the majority, in which the Roentgen features render tuberculosis improbable. For example, a young man for four years had occasional fever, spat up large quantities of purulent sputum, and spent several years in tuberculosis sanatoria. The unusual shadow in his right upper lobe, so unlike tuberculosis, (fig. 164,) immediately cast doubt on the diagnosis. At operation, a large abscess cavity, surrounded by dense connective tissue, was found.

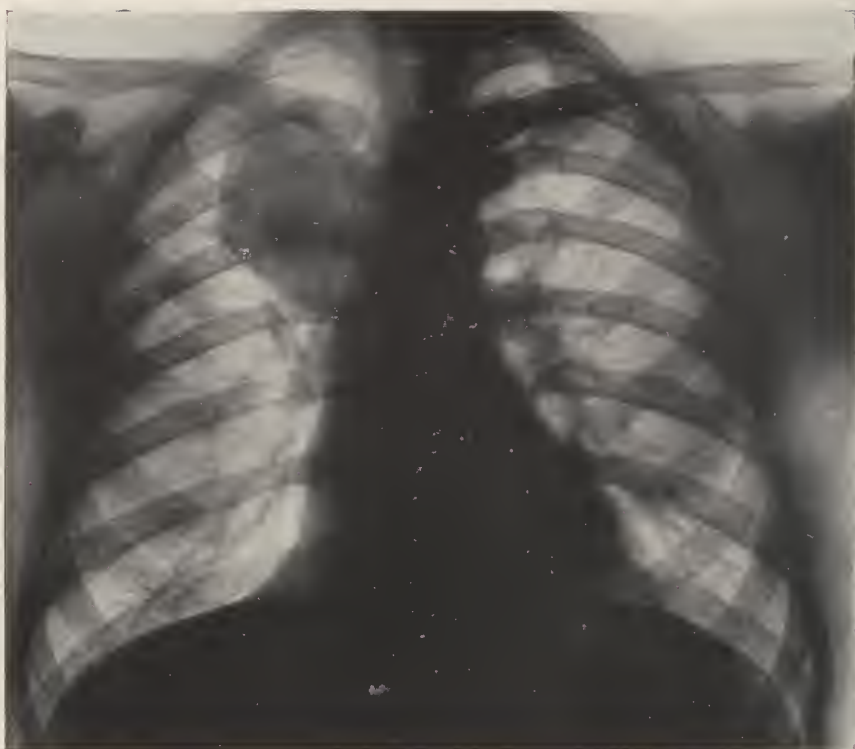


FIGURE 164

Chronic abscess of right upper lobe. At operation a large abscess cavity was found within the indurated area represented by the shadow.

THE HEALING OF ABSCESSSES

The Roentgen study of acute abscess and gangrene may furnish us objective evidence of the progress of the disease to a complete cure. It must be clear from the nature of the inflammatory process that this can most readily take place in the early period of the disease before fixed tissue changes have occurred in the lung. We find in fact, that a spontaneous cure results in approximately one-third of the cases of acute abscess and gangrene, practically always within three months of their onset. The Roentgen evidence of such a cure will consist in a complete disappearance of the cavity and its surrounding infiltration. To illustrate, the abscess in the right upper lobe (fig. 165) was of about two weeks' duration and was acquired presumably by aspiration during alcoholic coma. Within one month, cough and expectoration

ceased and the patient was completely restored to health. The only vestige of the abscess which was then to be found on the plate was a few coarse strands of scar tissue at its previous site. (Fig. 166.)



FIGURE 165

Abscess cavity in right upper lobe (arrow).
two weeks' duration.

FIGURE 166

Same case one month later. Abscess healed,
leaving only a few strands of fibrous tissue.
(Arrow.)

Even considerably larger cavities may disappear in the course of a few weeks. Thus, no remains of the large cavity seen in figure 155 could be found on the plate taken three weeks later. This capacity for healing defects so promptly and completely is characteristic of the lung and it depends on its unique spongy structure. Acute abscesses do not heal slowly by granulation as is the rule elsewhere in the body. They are on the contrary, quickly obliterated by the expansion of the surrounding lung when the exudate which fills the alveoli is absorbed and they are once more distended with air. This occurs as soon as the gangrenous wall of the cavity, which has maintained the surrounding lung in a state of consolidation, is sloughed out and expectorated. In the chronic abscess cavities on the other hand, where a rigid fibrous wall has been formed, such a rapid healing of the cavity is precluded; in fact these cavities, with rare exceptions, cannot heal spontaneously.

When the progress of a lung abscess is thus observed on the Roentgen plate, we are forced to the conclusion that little or no dependence can be placed on the disappearance of the symptoms or the physical signs, as a proof of the patient's cure. It is not uncommon, a few weeks or months after the onset of the disease, for the patient to experience a great improvement; the cough, expectoration and fever may entirely

disappear and both patient and physician congratulate themselves on the happy issue of a dreadful malady. Nevertheless, when we make a careful Roentgen examination there will be found in a number of these cases, an apparently insignificant residue of infiltration in the lung. If, as may happen, this focus of disease contains the germs of anaerobic infection, the latter may lie dormant for some time until they later become the starting point for a renewed infection of the lung.

The observer who is disposed to disregard such residual foci will profit by an experience such as the following:

A young woman acquired a postoperative abscess of her right lower lobe. After two months of the usual symptoms she improved to such a degree that she was unwilling to submit to further treatment and left the hospital. There were no abnormal physical signs in the chest and the bronchoscopic examination revealed only a slight dilatation of the right lower bronchus, from which issued a minimal amount of secretion. On discharge from the hospital her plate showed a small pneumonic area in the middle of the right lower lobe. (Fig. 167.) Witness the unfortunate outcome of this case. Three months later the patient appeared with a recurrence, in an aggravated form, of all her earlier symptoms, including large pulmonary hemorrhages and high fever. The plate then made, (fig. 168.) showed an extensive infiltration in the upper part of the right lower lobe, at the summit of which a small cavity is to be seen.



FIGURE 167

Residual infiltration in right lower lobe (arrow.) Two months after a postoperative abscess of lung. Clinical cure.



FIGURE 168

Same case three months later with a recurrence of symptoms and an extensive suppurative process in right lower lobe.

The significance of even vague shadows on the plate of patients who are known to have had a lung abscess and in whom symptoms which may be attributed to a simple bronchitis persist, cannot be overestimated. These shadows acquire an unwonted importance when we view them in retrospect and realize with what ominous possibilities

they were freighted. In figure 169, only on close examination can we discern in the right upper lobe a small annular area of infiltration and a few coarse shadows near the root of the right lung. The patient traced his illness back seven months when he contracted influenza and pneumonia. This was followed by a persistence of the fever and

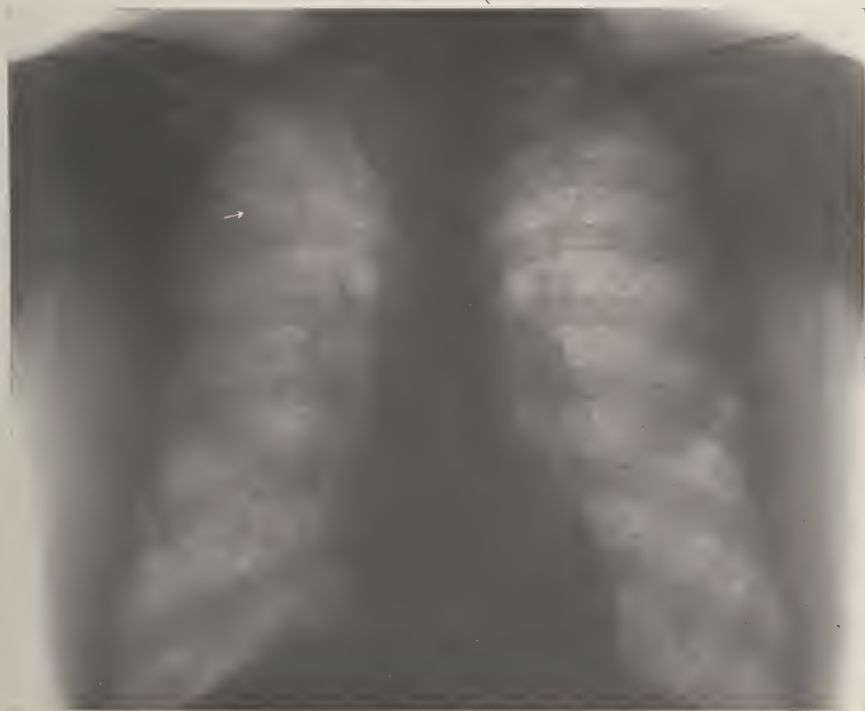


FIGURE 169

Residual infiltration after postinfluenzal abscess of seven months duration. (arrow.)
See figure 170.

expectoration which gradually became purulent and fetid. These symptoms continued until the time of the examination. His only signs consisted of dullness and diminished breathing in the right infra-spinous region. The clinician, were he ignorant of the previous history of the patient might be pardoned for doubting the adequacy of these few Roentgen shadows to explain a persisting suppuration. Yet the sequel proved this to be so. A plate, (fig. 170,) made eight months later, revealed a fluid-containing cavity, which developed at the site of the previous infiltration. A residual necrotic focus, for a long time lying dormant and causing only a bronchitis, had reinfected the lung about it and brought in its train the outspoken symptoms of lung suppuration.

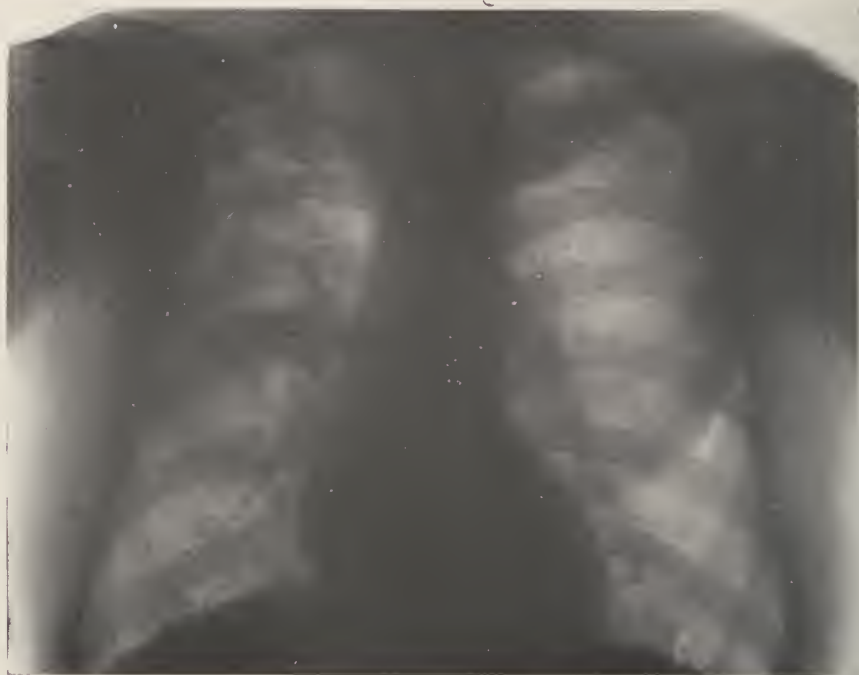


FIGURE 170

Same patient as in Figure 169 eight months later. Extension of infiltration with abscess formation.

Such illustrative cases could be multiplied to emphasize the need of conservatism in pronouncing a gangrenous lung abscess cured when it has lasted more than a few months. As we have seen, relief from symptoms cannot be accepted as an index of cure and equally little weight should be attached to an absence of physical signs. No one who has had any experience with these cases can fail to be impressed with the meagreness of the physical signs in many cases of suppurative lung disease. Even large areas of infiltration may produce only slight dullness and diminished breathing. The smaller residual foci, especially those at the roots of the lungs, which play so important a role in the exacerbation of the process, are nearly always latent and only the Roentgen examination brings them to light.

ABSCESS CAVITIES

Abscess cavities, because they are the most striking manifestation of lung suppuration, may properly lay claim to a more detailed consideration. Although cavities are invariably to be found in the lungs at autopsy or operation, in only a small percentage of the cases can their presence be determined by the physical examination. This percentage

is notably increased when we resort to the Roentgen-Ray, yet even with this aid they entirely elude us in at least one-half of the cases. Thus in one hundred cases of lung suppuration in which this feature was investigated, there were indubitable physical signs of a cavity in only twelve, where excavation of the lung was demonstrated on the plate in fifty.

Various reasons may be assigned for this circumstance. Cavities which are full of secretion, especially when they are small, may be entirely obscured by the infiltrated lung about them. If they are later emptied by expectoration, they may come to light as circular defects within the consolidated area. Thus, the lung abscess in the right upper lobe in figure 171 revealed on the plate only a dense pneumonic consolidation. Yet, two days later, after an unusually profuse expectoration, the outlines of several small cavities appeared. (Fig. 172.) In

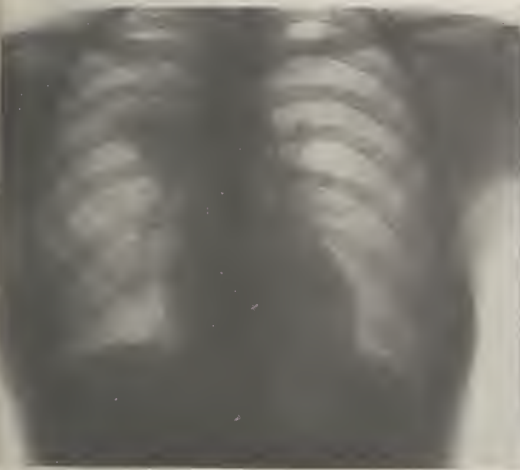


FIGURE 171

Lung abscess, right upper lobe. Cavities not seen.



FIGURE 172

After profuse expectoration, several small cavities are visible.

truth, the Roentgen shadows, in whose lineaments we attempt to trace the pathology of this disease, represent but an imperfect image or silhouette of the affected lung. The limitations of the examination cannot be better exemplified than by the following series of three plates of a patient taken at short intervals. Figure 173 portrays the area of suppuration on admission to the hospital. No cavity was visible. When again examined, a few weeks later, a large cavity was noted on the plate. (Fig. 174.) But it would be a mistaken view to regard this as



FIGURE 173

Pneumonic consolidation at root of lung.
No abscess cavity seen.

FIGURE 174

Same case as figure 173, a few weeks later.
A large cavity now visible.
See figure 175.

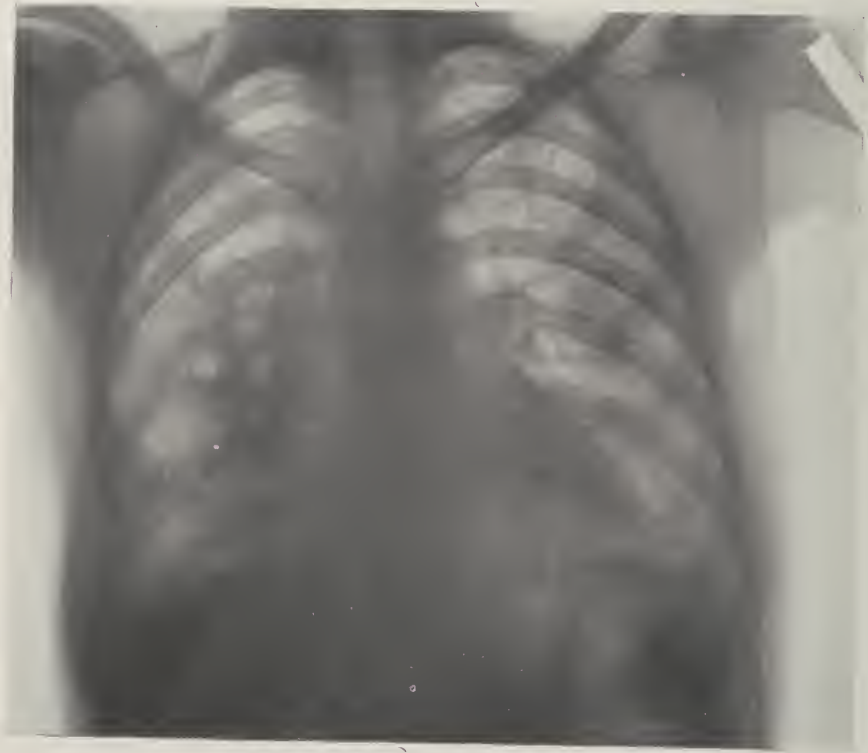


FIGURE 175

Same case as figure 174 after partial resection of lung. Shows a number of cavities previously hidden by consolidated lung.

the extent of the necrotic process. A short time after, the patient was operated on and a considerable portion of the diseased upper lobe was resected. There were then visible five cavities of different sizes which had been entirely obscured by the overlying lung. (Fig. 175.) Finally, when at autopsy it was possible to examine the lung in detail, it was found to be full of innumerable small and large necrotic abscess cavities.

At times the Roentgenologist must vary his procedure in order to detect cavities which are located in unusual situations. In figure 176 is illustrated an irregular cavity which lies within a diseased middle lobe. It was necessary to resort to the oblique position in order to disclose this cavity, as it was completely obscured by the heart in the dorso-ventral position. The same difficulty was encountered to a lesser degree

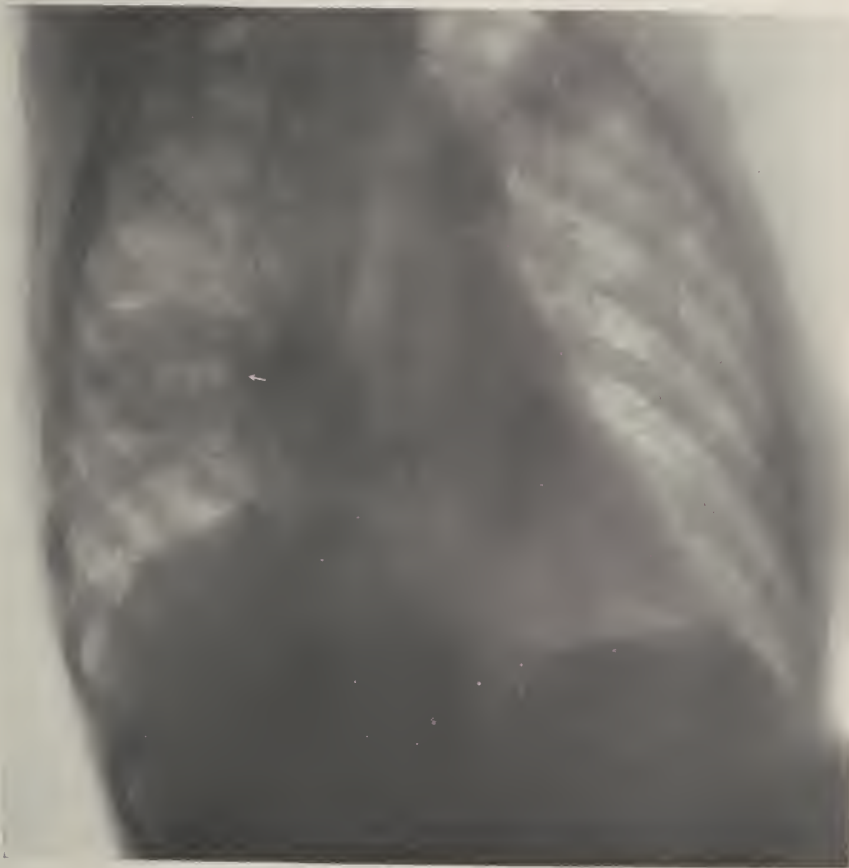


FIGURE 176

Consolidation of middle lobe, with irregular abscess cavity, twenty-two months after tonsillectomy. Cavity only seen by oblique examination as it was situated behind the heart.

in another case of lower lobe abscess on the left side. (Fig. 177.) Here again, the cavity was partly hidden by the left ventricle in such a way that the fluid level was incomplete and doubt arose as to the nature of the shadow. The patient was re-examined in the recumbent position, lying on his right side. By this manoeuvre the fluid level was shifted clear of the heart shadow and all doubt as to the existence of a cavity was removed. (Fig. 178.)



FIGURE 177

Abscess of left lower lobe, partly obscured by the left ventricle.



FIGURE 178

Same case as figure 177, in lateral recumbent position showing change in level of fluid in cavity.

The Roentgen demonstration of larger cavities seems often to be facilitated by an increased density of the lung about them. This phenomenon has been observed in cases associated with pneumothorax. A cavity which may not have been previously visible may be clearly outlined within the compressed lung after the entrance of air into the pleural cavity and in doubtful cases this procedure may be resorted to for purposes of diagnosis.

In general, abscess cavities are circular and smooth in outline; occasionally, and this is true of the smaller ones, they may be oval or irregular. They are rarely surrounded by a recognizable capsule but appear rather as defects within the pneumonic infiltration which surrounds them. Cavities which have attained a moderate size practically always communicate with a bronchus; the free access of air causes their fluid contents to assume a horizontal level which is surmounted by a hemi-spherical air space. Such a fluid collection, which can be caused to shift its level by a change of position, is so characteristic that it

constitutes positive evidence of a cavity, even when, as frequently happens, the wells of the latter are indistinct or invisible. Cavities vary in size from some which are barely distinguishable to others which occupy a whole lobe. The larger cavities are found with much greater frequency in the acute aspiration abscesses than in the chronic pneumonic forms. This finding is in harmony with the greater role which the gangrenous process plays in the former. The rapidity with which abscess cavities may form is astounding. In virulent cases we have observed on the plate the necrosis and complete excavation of a lower lobe within forty-eight hours and have verified this by autopsy.

LOCATION OF THE ABSCESS

Suppurative disease may affect any lobe of the lung and in the longer standing cases a multiple lobe involvement is frequent. If we analyze a sufficiently large number of cases in respect to the localization of the disease, a distinct difference will be found to exist between the acute aspiration or post-operative abscess and the chronic pneumonic type. In the former there is a predilection for the upper lobes, which are involved twice as frequently as are the lower lobes. In only two instances have we encountered isolated middle lobe suppuration, one of which, figure 176, followed tonsillectomy, the other developing insidiously after a pneumonia. The diagnosis of these cases of middle lobe disease presents unusual difficulties to the physical examiner because of the central location of the morbid process, whose physical signs are notoriously meagre.



FIGURE 179

Abscess involving contiguous portions of right upper, middle and lower lobes. Cavity with fluid level.



FIGURE 180

Same case as Figure 179 after removal of entire right lung.

The exact localization of the abscess, which we owe in such full measure to the Roentgen examination has a practical bearing which transcends its value as a mere method of diagnosis. To the surgeon who contemplates the drainage of a cavity or the incision of a lobe, the situation of the abscess will determine his method of approach and for this purpose an accurate localization is essential. Even with the Roentgen examination this is not always possible. Especially on the right side, the overlapping of the three lobes in the middle third of the chest will render it difficult to assign an infiltration in this region to any one lobe or to the adjacent portions of all three lobes. Here stereoscopic examination may be of service. In figure 179, for example, it is impossible to deduce from the plate alone, whether the abscess is situated in one or more lobes; at operation a considerable portion of all three lobes was found to be diseased. In these cases, dependence must be placed on the location of the physical signs to distinguish between an affection of the lower part of the upper lobe and the upper part of the lower lobe, the shadows of which will be super-imposed on the plate.

COMPLICATIONS OF LUNG ABSCESS

During the clinical course of a lung abscess the patient is constantly threatened with serious complications. As some of these involve the lungs and pleura, they may well engage our attention. Perhaps the commonest complication of lung abscess is a pleural effusion, which may be due to the rupture of a cortical abscess into the pleural cavity. If, as is usually the case, there is a communication with a bronchus, a pyo-pneumothorax results with all its attendant symptoms. The complete picture of the abscess with the associated effusion may then be visible on the plate and the entire sequence of events may be reconstructed from it. As an example we may instance the following:

A middle-aged man contracted pneumonia which in five weeks assumed the typical character of lung abscess. Increasing dyspnoea and fever brought him to the hospital where the Roentgen examination revealed a pneumonic consolidation in the mesial portion of the right upper lobe and a collection of fluid and air at the right base. (Fig. 181.) Above the latter the lung was partly attached to the chest wall by adhesions.

The slow approach of an abscess toward the pleural surface of the lung may permit the formation of adhesions between the parietal and visceral surfaces of the pleura before it perforates. When this finally occurs, the pyo-pneumothorax which develops may be entirely encapsulated and a unique Roentgen picture such as figure 277 may be the result. Here again, following an acute influenzal lung abscess with gangrene, perforation occurred on the posterior aspect of the lung. The adhesions about the pleural effusion, which are well shown on the plate, were found at operation to be firm and effectually walled the effusion off from the general pleural cavity.



FIGURE 181

Abscess in mesial part of right upper lobe with small cavity. Perforation of abscess with pyo-pneumothorax. Falciform shadow due to adherent lung.

A characteristic symptom which may arise when there is a free communication between the pleural cavity and a bronchus, consists in a paroxysm of coughing and the expectoration of a large amount of purulent sputum when the patient changes his position. If the opening of the pleuro-pulmonary fistula is situated above the level of the fluid, the latter will flow upward and be discharged through the fistula when the patient assumes the recumbent position. It may thus be possible to localize accurately the fistula by noting on the fluoroscopic screen at what position of the fluid level the patient begins to cough and to expectorate pus.

It is not necessary for an abscess actually to perforate the lung, in order to stimulate the pleura to inflammatory reaction. A nonpurulent effusion may thus result from a subpleural abscess and with an increasing intensity of the reaction we may observe the gradual change in the character of the fluid from a serous to a frankly purulent one. The problems which present themselves to the clinician in these

cases are complex and their solution is often facilitated by the Roentgen examination. This is especially true of some cases of chronic suppuration of the lung which have lasted for years and in which the symptoms of pulmonary disease may to a great extent have subsided. At any time, however, the process may be aroused to activity and if by chance the pleura is then infected from a cortical abscess, the resulting pleurisy may so dominate the clinical picture that it may on a cursory view appear to be the primary condition. When the chest of such a patient is aspirated it may happen that at one level clear fluid is obtained from the pleura and at a higher one, pure pus from an abscess cavity. The clinician is here under the necessity of distinguishing between an abscess with a secondary serous effusion in the pleural cavity and a bilocular empyema, which contains purulent fluid at one level and serous fluid at another. The Roentgen plate may supply the solution, as it did in the following case:



FIGURE 182

Lung abscess of four years' duration with secondary pleural effusion which obscures the abscess. Purulent fluid obtained from middle of chest and clear fluid at base. See figure 183.

The patient, a young man, had contacted a lung abscess four years previously, possibly from the aspiration of a foreign body. Following its insidious onset his disease became almost quiescent until the recent development of a pleural effusion. Figure 182 shows the Roentgen appearance of his chest at the first examination. When his left chest was aspirated at the base, clear, polynuclear fluid was withdrawn, whereas insertion of the needle higher up in his axilla, brought forth pure pus. The latter issued from a walled-off abscess cavity which had been entirely obscured by the pleural effusion. After the removal of the latter, a plate made in the lateral recumbent position clearly revealed this cavity and its purulent contents. (Fig. 183.)



FIGURE 183

Same case as figure 182 after removal of pleural effusion. Shows a large abscess cavity with purulent contents. Shifting fluid level in lateral recumbent position.

We must especially keep in mind the possibility of latent suppuration of the lung in the case of children who, with no adequate antecedent disease, develop encapsulated effusions. As the basis of the suppuration there may exist a foreign body which has been aspirated into a bronchus, of which everyone, including the patient, may be ignorant. The cough, lasting for many months may be attributed to a bronchitis

until the rupture of a bronchiectatic abscess and the resulting empyema lead to a closer scrutiny of the case and the discovery of the underlying infection of the lung. (Fig. 184.)

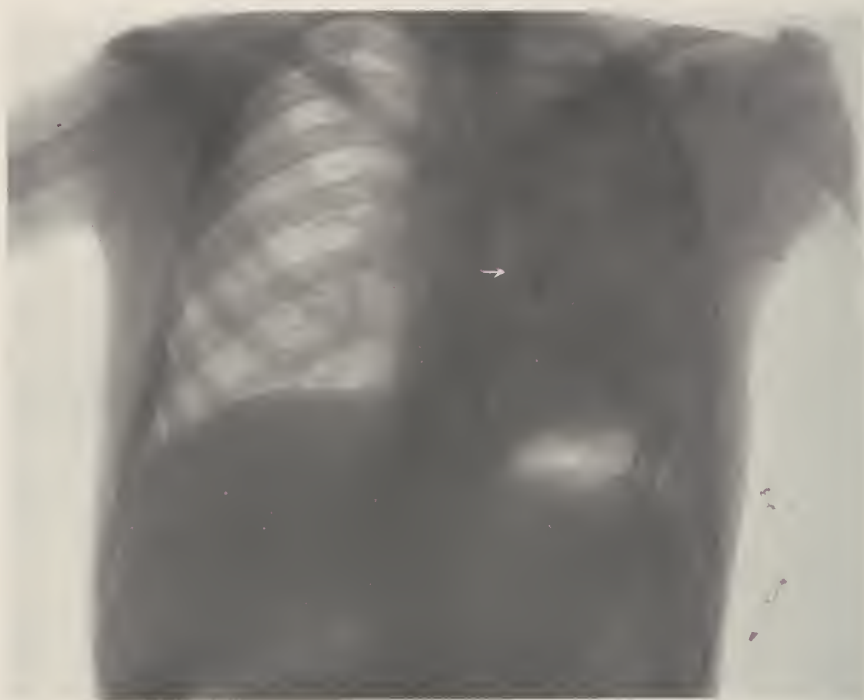


FIGURE 184

Chronic suppuration of lung in a child following aspiration of foreign body. First sign of acute disease resulted from perforation of a cortical abscess with empyema. Arrow points to piece of metal in bronchus.

Although the pleural complications of lung abscess are usually of serious import and often add to the difficulties of operation, there are cases in which they appear to exert a favorable influence on the outcome. Thus in acute abscesses, especially those following influenza the early rupture of a cortical focus with the resulting empyema, has often led to prompt drainage of the suppurating lung and a rapid cure of the patient.

Only casual reference is necessary to embolic abscesses of the lung which are secondary to septic thrombosis of the systemic veins. The small abscesses, which are usually multiple and produce a furunculosis of the lung play but a minor part in the clinical picture. On the Roentgen plate they cast smaller or larger irregular circular shadows which are indistinguishable from a lobular pneumonia.

Although in the vast majority of cases of lung abscess, especially of the acute variety, suppuration is associated with gangrene, there are occasional cases, usually following lobar pneumonia, in which resolution is complicated by simple suppuration without gangrene. The pulmonary cavity, which is easily seen on the plate, does not communicate with a bronchus and the disease may run its course to complete healing without the expectoration of pus.

We have attempted to show in our discussion of lung suppuration, that the Roentgen examination is qualified to supply the clinician with valuable information in regard to the diagnosis and prognosis of this disease and also at times to point the way to intelligent treatment. One of the Roentgen features of lung suppuration, the abscess cavity, is sufficiently typical to warrant an immediate diagnosis; on the other hand, we must be prepared, in the numerous instances in which it is absent, to make the diagnosis without it and to interpret correctly the

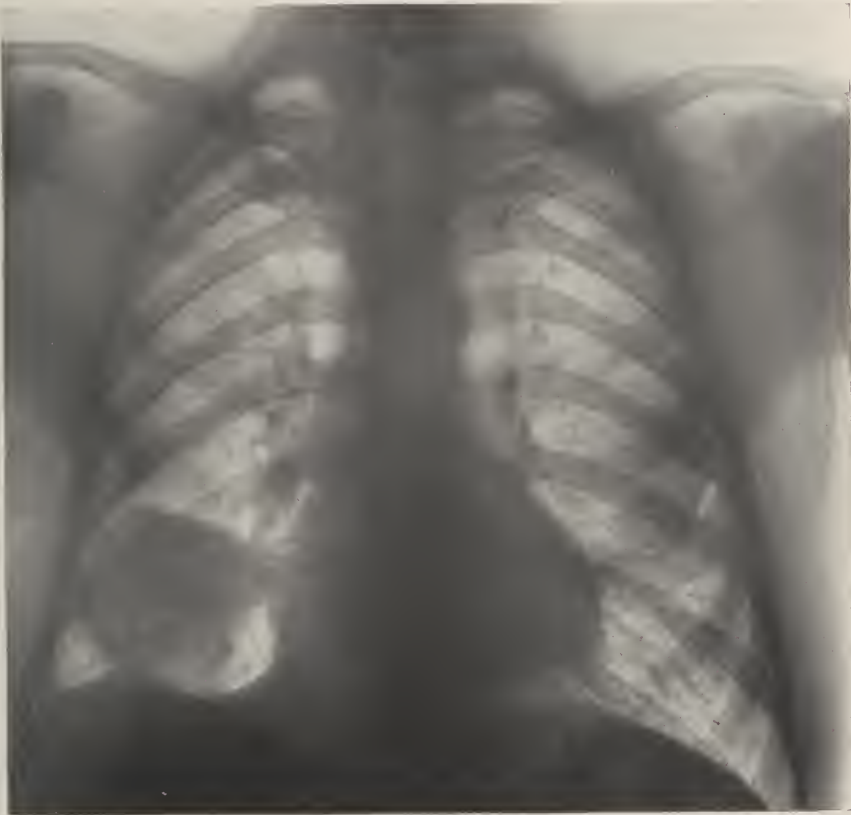


FIGURE 185

Chronic postpneumonic lung abscess. Shadow has circular shape and definition of a lung tumor.

less characteristic infiltrations which are nevertheless an essential element in the disease. It will be necessary at times to distinguish these from tuberculosis, new-growths (fig. 185) and even encapsulated effusions, which is not always an easy matter. As an isolated method of examination there are definite limits to the application of the Roentgen ray in cases of lung abscess; regard for these limitations will impel the examiner constantly to turn to the patient himself for confirmation of his interpretations. The Roentgenologist left to his own resources may therefore frequently be at a loss, unless he summons to his aid such significant facts of the clinical history as a previous operation or a pneumonia. It must also be evident that the expectoration of fetid sputum and the finding in it of elastic fibres and clubbing of the fingers will exert a determining influence on the interpretation of questionable shadows. As in other thoracic diseases, the Roentgen examination has its greatest usefulness in the diagnosis of lung suppuration when its findings are made a co-ordinate part of the whole clinical picture and are brought into harmony with it.

CHAPTER IX

Non-Tuberculous Chronic Infections of the Lungs

Aside from tuberculosis, which accounts for the vast majority of cases of chronic lung infection there is a not inconsiderable number of cases in which other agencies than the tubercle bacilli are responsible for chronic pulmonary disease. Among these we find the ordinary pathogenic bacteria of acute pneumonia which under favorable conditions may incite a more persistent inflammatory process and such rarer causes as the virus of syphilis and the various mycotic organisms. Of these cases, chronic indurative pneumonia constitutes the great majority, whereas the other forms of pulmonary disease are exceptional.

CHRONIC INDURATIVE PNEUMONIA

Clinically the chronic pneumonias constitute an ill-defined group of cases of widely varying symptomatology. Their insidious development and their frequent similarity to pulmonary tuberculosis often lead to their confusion with this disease, while in other cases the presence of particular symptoms, such as frequently recurring hemorrhages, may arouse a strong suspicion of new-growth. This variable clinical picture is dependent on the peculiar pathology of the disease as it passes from the stage of persisting pneumonia through different degrees of organization and induration to fibrosis. In the course of this organization, secondary changes such as bronchial dilatation and abscess formation are not uncommonly produced, which impress their characteristic symptoms on the clinical picture.

The commonest form of indurative pneumonia is found in older individuals and frequently follows an acute basal pneumonia. Of greatest interest are the cases of the "grippe" type in which the symptoms and signs of pulmonary inflammation are so slight that they are not recognized. Frequently only the persistence of cough and expectoration and the almost invariable spitting of blood with slight temperature indicate the existence of pulmonary disease which, often as not, is looked upon as pulmonary tuberculosis.

The Roentgen picture in these patients commonly reveals a shadow at the base of the lungs, which in the one case may have the density of a consolidation, whereas in the other it has the faintness of a resolving process. The basal situation of the process at once serves to distinguish these cases from tuberculosis.

Marked structural changes in the lung may be induced in the sub-acute stage of indurative pneumonia, which are reflected in striking Roentgen shadows. Thus, the massive consolidation seen in figure 133 was found at operation, when the lobe was excised, to be due to a sub-acute organizing pneumonia, with beginning bronchial dilatation and purulent bronchitis. The illness of the patient dated from a pneumonia contracted seven months before and at the time of operation the disease acquired an acute character.

As the subacute stage of the disease is passed and with an increasing tendency to fibrosis, we may at times observe on the Roentgen plate an increasing density and coarseness of the shadows. At this point the final direction which the pathological process will take is determined by a number of secondary factors. In certain cases there develops a general bronchial dilatation to which, especially in older individuals, there is super-added an anaerobic infection. The wall of a bronchus becomes gangrenous and the complete clinical picture of suppuration of the lung is evolved. Thus in figure 157 are seen the coarse shadows of an organizing pneumonia of eight months' duration which terminated in gangrene of the lung. Although abscess cavities originating in the dilated bronchi are invariably found in the excised lungs, they are rarely visible on the plates as they are imbedded in and obscured by the indurated lung.

In the absence of anaerobic infection the disease is apt to remain a mild one and to continue for years with few or no serious symptoms. These are the cases which we have already described in the chapter on bronchial diseases, as multiple bronchiectases, which are characterized by a profuse expectoration and the almost universal presence of clubbed fingers. The disease here most often involves the lower lobes.

Of greater interest, because of the diagnostic problem presented, are the occasional cases of chronic pneumonia with bronchiectatic formation which involve also the upper lobes. The history of these cases extends usually over so long a period that the symptoms attending their onset have passed from the memory of the patients, who only know that they have been coughing for years. In many instances the disease has been regarded as tuberculous and the patients have been inmates of tuberculosis sanatoria. For this belief there is often much justification, as the physical signs, the profuse expectoration and the occasional hemoptyses would lend credence to such a view. The constant absence of tubercle bacilli and the remarkably good general health of these patients sooner or later shakes the confidence of the physician in the diagnosis of tuberculosis, which the Roentgen examination makes even more doubtful. The plate in these patients commonly shows unilateral fibrosis of

the lungs in which numerous thin-walled cavities may be seen. (Fig. 186.) There is also usually a retraction of the chest wall and a displacement of the thoracic viscera toward the diseased side.



FIGURE 186

Chronic indurative pneumonia with multiple bronchial dilatations. Displacement of heart and mediastinum.

In distinguishing these cases from tuberculosis, the absolute limitation of the process to one lung is of the greatest importance. While fibroid tuberculosis may be restricted to one lung, usually, if the case is of some duration, the Roentgen examination will disclose some degree of infiltration of the opposite lung.

From a practical therapeutic point of view much interest attaches to chronic disease of the lung incident to the obstruction of a bronchus by a foreign body. The partial or complete occlusion of a bronchus,

whether by an external tumor or aneurysm or by an intrabronchial growth or foreign body, may result in a stasis of bronchial secretion with subsequent infection which, if unrelieved, may terminate in subacute or chronic indurative pneumonia. Provided the obstruction is removed in time, as in the case illustrated in figure 19, permanent damage to the lung may be obviated. In case of continuing occlusion however, the damage to the lung will be permanent, leading to greater and greater fibrosis. In chronic pulmonary disease in children one must especially have in mind the possibility of the aspiration of a foreign body into a bronchus. This may happen without the child being aware of it and it becomes the starting point for a long train of symptoms characterized by fever, cough, profuse expectoration and ultimately clubbing of the fingers. The Roentgen examination usually shows extensive pulmonary shadows, pneumonic in type and at times the shadow of the foreign body. (Fig. 184.) In this patient who, probably four years before, aspirated a portion of a tin whistle, serious attention was only drawn to the pulmonary condition by the sudden perforation of a cortical bronchiectatic abscess with empyema. Unfortunately, after such a long duration, removal of the foreign body and drainage of the pleural cavity had little or no influence on the chronic lung disease.

In the types of induration which we have thus far considered, the lung is solely involved, to the exclusion of any disease of the pleura. There is, however, a group of cases in which the fibrosis may be described as pleuro-pneumonic in that, either primarily or secondarily, the pleura shares in the fibrotic process. In the complete clinical picture of pleuro-pulmonary fibrosis, it may be a matter of some difficulty to determine the priority of the process in the lung or in the pleura. There are undoubtedly some cases in which the inflammatory process is initially a pleuro-pneumonia in which the fibrosis progresses simultaneously in the lung and pleura. In these cases, with or without pulmonary symptoms, there is a progressive shrinkage of the chest with a marked thoracic deformity and cardiac displacement and an obliteration of the pleural cavity.

On the Roentgen plate these changes are strikingly in evidence. As seen in figure 187, the pleura is greatly thickened so that the details of the underlying lung are obscured and the heart and mediastinum show an extreme displacement. In the physical examination of these patients the clinician is placed at a great disadvantage. The thickened pleura usually does not permit the transmission of breath sounds; the displacement of the trachea to the side of the sternum, by the production of pseudoamphoric breathing, simulating a cavity, leads to a diagnosis of pulmonary tuberculosis.



FIGURE 187

Chronic non-tuberculous disease of lung and pleura. Pleuro-pulmonary fibrosis. Deformity of chest and displacement of heart and mediastinum.

The cases of pleuro-pulmonary fibrosis which begin in the pleura are probably the sequel of untreated empyemata. Often they have been incompletely evacuated, leaving behind a residuum of inflammatory material which progresses to organization. In some cases the pleural exudate perforates the lung which it infects and involves in the inflammatory process; in other cases, fibrosis invades the lung along the interlobular septa. In either case the clinical and Roentgen picture of pleuro-pneumonic induration which we have described, ultimately develops.

The many-sided clinical picture which chronic indurative pneumonia may exhibit is nowhere better illustrated than in the cases which simulate a new growth of the lungs. Beginning insiduously as a pneumonia, the pulmonary infiltrations may progress to carnification in six or eight months. In addition to the usual symptoms of the infection, a persistent, almost daily hemoptysis and thoracic pain may be present. Such a train of symptoms, especially if there is some loss of weight, cannot fail to arouse the suspicion of a new growth of the lung. The Roentgen examination in some of these cases may even add to the diffi-

culties in diagnosis as the type of shadow found may easily be confused with that of a tumor. The area of induration in the lung may be sharply circumscribed and have the density and contour of a malignant growth. Thus in figure 188 is seen a dense, tumor-like shadow which was found



FIGURE 188

Organizing pneumonia. Both clinical and Roentgen picture that of a tumor of the lung.

six months after a lobar pneumonia and which was presumed to be due to a neoplasm. At operation, however, a carnified lung was found. If these patients are observed for a short period and repeated Roentgen examinations are made, it will be discovered that the size of the infiltration is subject to an alternate rapid increase and decrease in size which is not consistent with a tumor growth.

It will be evident that in the diagnosis of a disease which has so protean a clinical picture and Roentgen appearance as chronic indurative pneumonia, a knowledge of the chronology of the symptoms and their sequence is of the greatest moment to the examiner. The definite history of a pneumonia or of antecedent pleural disease or of the aspiration of a foreign body may point clearly to the true nature of the disease.

In the interpretation of the extensive unilateral infiltrations and bronchiectases the clinical examination is of major importance. The discovery of clubbed fingers within a few months after the onset of the disease is of the greatest significance, as they develop very rapidly in non-tuberculous infections, whereas it requires years for them to make their appearance in cases of tuberculosis. Finally, in occasional cases of this type also a positive Wasserman test or the discovery of actinomycosis in the sputum will place an entirely novel interpretation on the Roentgen shadows.

CHAPTER X

Actinomycosis of the Lungs

Actinomycosis is one of the rarer causes of chronic non-tuberculous disease of the lung. The nature of the inflammatory process, in which suppuration and induration of the lung are combined with bronchial dilatation and abscess formation makes for a Roentgen shadow of great density and usually of considerable extent in the involved lung. In interpreting these shadows we must keep in mind the tendency for actinomycosis rapidly to invade the pleura which becomes thickened and also to infiltrate and finally to perforate the chest wall. The dense shadows which we see when the disease is fully developed are therefore as much due to inflammatory thickening of the chest wall and pleura as to the pulmonary infiltration itself. Thus the dense shadow in the left upper lobe region in figure 189 was found in a patient who for some months had the symptoms of suppurative lung disease. Gradually a prominence of the left upper chest began to form until, at the time of the examination, there was a red fluctuating mass over the left upper



FIGURE 189
Actinomycosis of lung, pleura and chest wall.

chest. This was incised, disclosing an induration of the chest wall communicating by tortuous sinuses with the pleural cavity, where considerable pus containing the ray fungus was found. The lung beneath was so indurated that after the evacuation of the pus, the Roentgen shadow was of the same density as before. A similar case is illustrated in figure 190.



FIGURE 190

Actinomycosis of lung and pleura

It will be seen that the Roentgen picture by itself presents little that is characteristic. The shadows are those of an indurative pleuropneumonia, such as may be found in other forms of lung suppuration. If, however, with such a shadow, there is associated a prominence and infiltration of the chest wall, the Roentgenologist may with some assurance infer that it is due to actinomycotic infection of the lung.

CHAPTER XI

Echinococcus Cyst of the Lung

It is the frequent experience of the Roentgenologist that diseases of the chest which are most difficult of diagnosis by the conventional methods are often most easily discovered by the Roentgen Ray. Nowhere is this more strikingly illustrated than in the case of echinococcus cysts of the lungs. Situated deeply in the parenchyma of the lung and having no communication with the bronchial tubes, they often remain clinically latent during the period of their greatest growth. It therefore happens that echinococcus cysts are accidentally discovered when their existence is least expected and the clinician is amazed that such large masses in the lung are consistent with a total absence of physical signs.

The two cases of echinococcus cyst of the lung which we herewith illustrate exemplify these features of the disease. The first, (fig. 191)

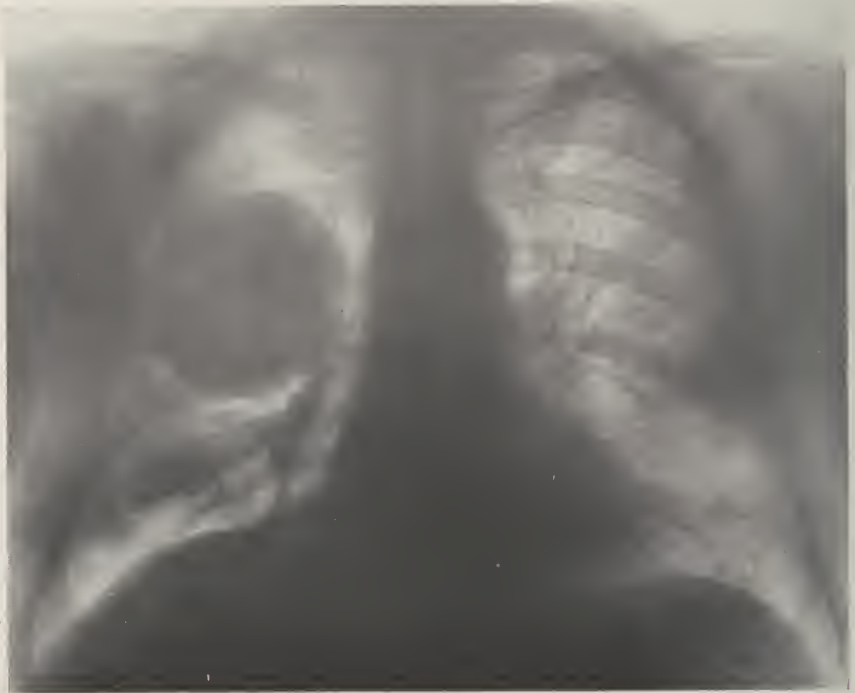


FIGURE 191

Solitary echinococcus cyst of the lung.

is a solitary cyst of large size in the right lung of a man of 55 years. His symptom was precordial and right thoracic pain which naturally directed attention to the heart and coronary arteries. There were no abnormal physical signs.

The second case, (fig. 192), shows multiple cysts of different sizes. Here also the patient, a young man who had spent some years in the Orient, had no physical signs of thoracic disease and the cysts were symptomatically latent. They were discovered incidentally to a gastrointestinal Roentgen examination.



FIGURE 192

Multiple echinococcus cysts. One cyst partly seen above the left diaphragm.

The Roentgen appearance of echinococcus cysts is so typical as to offer no difficulty in diagnosis. They are uniformly circular or slightly elliptical. The shadow is homogeneous, of considerable density and is sharply defined from the surrounding lung. No other type of pulmonary shadow excepting that of metastatic tumors, even remotely resembles it. However, pulmonary tumors which have attained the size of a cyst are rarely so uniformly circular nor so dense; moreover, the primary tumor will usually have made itself manifest in some other part of the body.

CHAPTER XII

Syphilis of the Lungs

The history of pulmonary syphilis as a clinical entity from the time of Pancritius, who popularized it, to the present day, illustrates the vagaries into which medical diagnosis may be led when it is subject to the enthusiasm or bias of the observer. In the pre-Wasserman era, the occasional discovery of a case of luetic disease of the lung only too often led to its promiscuous and unwarranted diagnosis. More recently the introduction of the Wasserman test and the recognition of the wide diffusion of syphilis have again given impetus to the diagnosis of pulmonary syphilis. Periodically such cases are reported for no other reason than the occurrence of physical signs of pulmonary disease in a syphilitic patient.

Yet it is common knowledge that syphilitic lesions are rarely found in the lungs at autopsy and therefore the clinical diagnosis of lung syphilis, even in the luetic patient, is always precarious.

These considerations apply with equal force to the Roentgen diagnosis of this disease. In view of the wide dissemination of tuberculosis it will not be an uncommon happening to find infiltrations in the lungs of patients who present clinical signs of lues or who exhibit only a positive Wasserman reaction. Nevertheless in spite of the overwhelming preponderance of tuberculosis as a cause of such infiltrations, these cases are periodically reported as examples of pulmonary syphilis. This practice is to be deprecated. A positive serum reaction should not of itself create a presumption in favor of syphilitic disease of the lung. On the contrary, if the Roentgen shadows have the features which we have learned to associate with tuberculous deposits, it is wiser, in view of the much greater frequency of the latter, to regard them as tuberculous. A suspicion of syphilis may be aroused by an atypical arrangement of the shadows or by some unusual circumstance of the case and the Roentgen and other data should then be so correlated as to be mutually confirmatory, if possible. In any case the diagnosis should be advanced with caution. It will gain probability in so far as other diseases can be excluded and it will attain certainty only by the therapeutic test, that is, by the disappearance of the infiltrations under antisyphilitic treatment.

From the Roentgen standpoint, because of the few cases which are encountered, no systematic description of the different forms of pulmonary syphilis can be attempted. We can however try to interpret the plates in terms of the various pathological processes which are known to occur in syphilitic disease of the lung. They are as follows:

(1) Gummata, (2) Interstitial pneumonitis, resulting in fibrosis, which probably arises from the peribronchial and perivascular connective tissue, (3) Diffuse, lobular syphilomatous infiltrations.

Having imposed on ourselves the limitations in diagnosis above indicated, a few cases of lung syphilis will here be recorded. Four of them responded to the therapeutic test and may therefore, be considered as proven. The other cases for various reasons to be indicated, could not be so tested and the diagnosis must therefore, remain in doubt. The clinical history however, taken with the Roentgen findings and the exclusion of other causes for the pulmonary changes, render the diagnosis at least probable.

Case 1. A middle aged man with symptoms of gastric ulcer had for some months been coughing and occasionally spitting blood. The Wasserman reaction was positive. Physical signs of dulness with some moist rales were made out at the right base posteriorly. The Roentgen examination made at this time showed the infiltration at the root seen in figure 193(a). The day after this examination the patient brought up

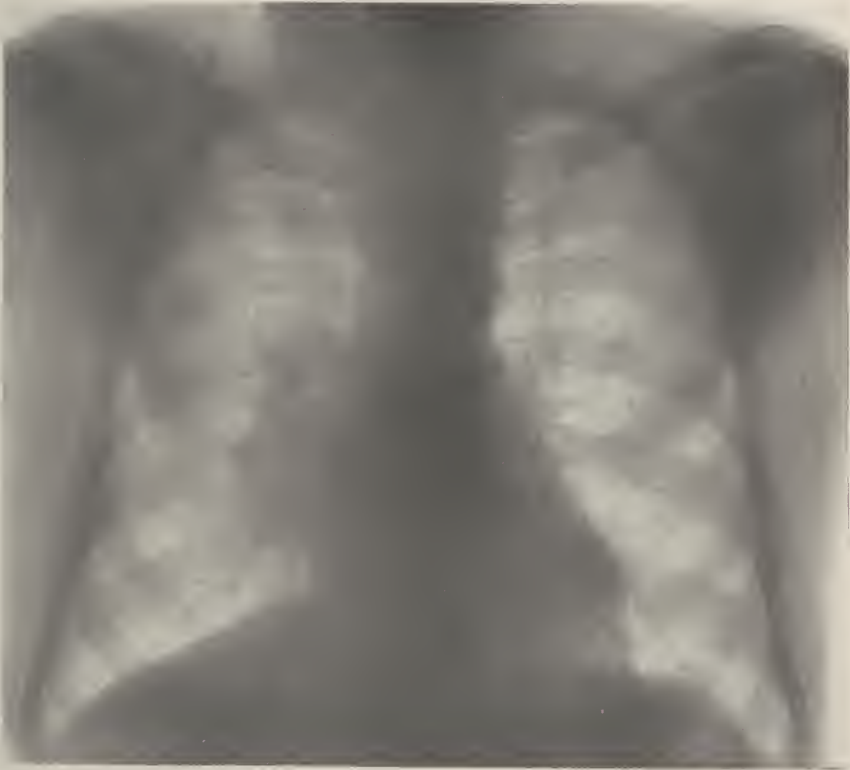


FIGURE 193(a)

Syphilitic infiltration at root of right lung. Clinical picture of lung suppuration. See figure 193.

fetid sputum in large amounts with some blood. The temperature ranged from 101° to 103° . An intensive course of antisyphilitic treatment was instituted with prompt relief of the symptoms. In three weeks the infiltration had entirely disappeared. (Fig. 193b.) Here we evi-



FIGURE 193(b)

Same case as figure 193(a). Disappearance of shadow at right root after antisyphilitic treatment.

dently had to deal with a syphilitic process, perhaps a gumma, which broke down and clinically presented the features of lung suppuration and gangrene.

Case 2. A woman, 63 years of age, for four months had a painless swelling of the sternal end of the left clavicle. The Wasserman reaction was strongly positive. Although she had no cough and was apparently in good health, the discovery of a few sibilant rales at the right base led to the Roentgen examination. This revealed the infiltration in the right lower lobe shown in figure 194. After a course of anti-

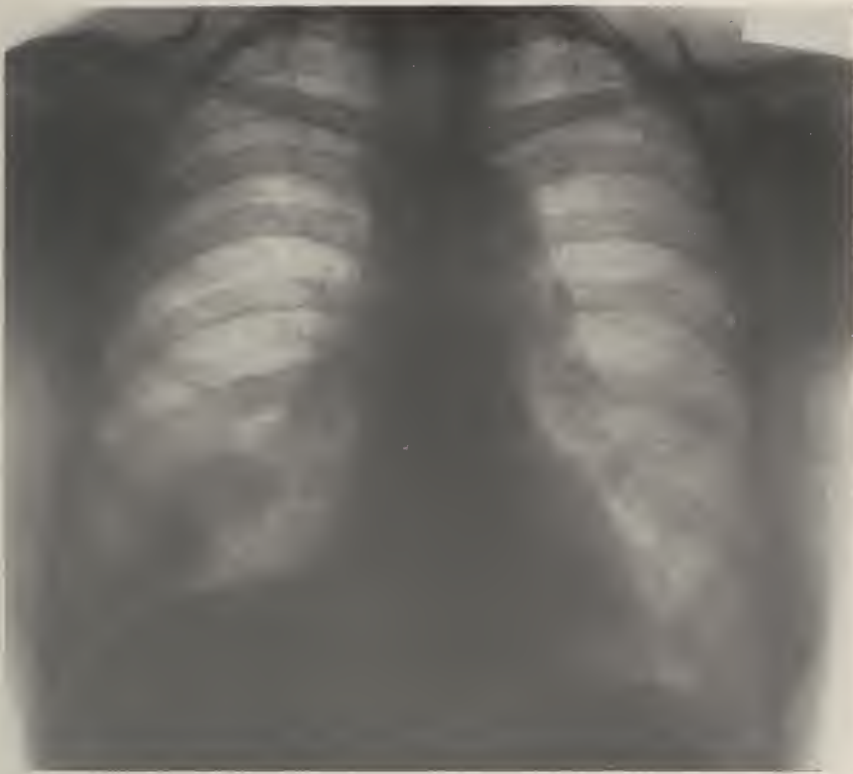


FIGURE 194

Pneumonic infiltration of right lower lobe in syphilitic patient. Process was latent. See figure 195.

syphilitic treatment, both the swelling of the clavicle and the infiltration in the lung disappeared. (Fig. 195.) Here we have illustrated a different type of syphilitic infiltration which manifested itself as a benign pneumonic process giving rise to no symptoms. The absence of fever or other symptoms of an acute inflammatory affection of the lungs and the prompt cure under treatment justify the diagnosis of lung syphilis.



FIGURE 195

Same case as figure 194. Disappearance of infiltration after anti-syphilitic treatment.

Case 3. This patient, a very anemic woman showed evidence of a severe degenerative kidney disease with marked albuminuria, associated with oedema of the lower extremities. In addition she had persistent oedema of the right arm. The Wasserman reaction was strongly positive. Roentgen examination of the chest revealed a circular shadow at the right apex, the periphery of which showed evidence of fibrosis. (Fig. 196.) Pulmonary physical signs were absent. The similarity of this shadow to that of a new-growth prompted a re-examination of the patient at intervals of some months. There was no increase in the size of the shadow. Owing to the renal condition, thorough antispecific treatment could not be instituted.



FIGURE 196

Probable gumma at right apex.

In this case it is presumed that the syphilitic lesion was a gumma of the upper lobe probably on or near the pleura which, by pressure on the subclavian vein accentuated an existing tendency to oedema. In spite of the positive Wasserman reaction, however, the diagnosis of syphilis was only ventured after other more common conditions, especially a neoplasm, were excluded as improbable.

Case 4. The following case, in its variegated features, illustrates the complexity of the clinical picture to which luetic lung disease may give rise. A woman of 38 years, who probably acquired syphilis from her husband a few years previously had moderate cough with mucopurulent expectoration for a few weeks. She then became acutely ill with high fever and copious expectoration. For a month before she entered the hospital she was thus very ill with daily fever, occasional chills, was much prostrated and became extremely emaciated. The examination of the lungs brought to light an unusual combination of physical signs. The left chest was immobile. The percussion note above was a dull tympany, whereas at the base there was flatness. The respiratory murmur was almost absent over the upper lobe; at the base the breathing was amphoric and accompanied by numerous musical rales, some of which were tinkling. Sputum was brought up in large amounts and was purulent, most of the pus cells being degenerated. There was evidently a consolidation of the lower lobe with an abscess cavity and atelectasis of the upper lobe.

The Roentgen plate, figure 197, shows a marked displacement of the trachea to the left. The absence of any retraction of the left chest indicates that this displacement is due to pulmonary atelectasis rather than to fibrosis, as the sequel showed. The entire left lung is consoli-



FIGURE 197

Syphilis of lung. At arrow is probably indicated a gumma of lung compressing left bronchus. (X) Symptoms of suppurative pneumonitis. Atelectasis of left upper lobe. See figure 198.

dated. At the origin of the left main bronchus there is an oval denser shadow of about the size of an olive. The patient was given vigorous antisyphilitic treatment to which she responded immediately. The fever subsided, the expectoration progressively diminished and within a few weeks all signs of consolidation and cavity disappeared. The condition of the lung four weeks after treatment was begun is shown in figure 198 where only a small residual infiltration, with a diaphragmatic adhesion is seen.



FIGURE 198

Same case as figure 197 after antisyphilitic treatment. Gumma has absorbed; residual infiltration at left base which subsequently disappeared.

It is not easy to interpret in pathological terms the nature of the inflammatory process in this case. We may venture to assume from the history, that there was a subacute, diffuse syphilitic process, perhaps

associated with gummata, which became necrotic. The entire absence of disease of the right lung is worthy of notice.

Case 5. Perhaps the most common form of pulmonary syphilis encountered, if it may at all be characterized as common, is the so-called syphilitic fibrosis. Pathologically this consists of broad connective tissue bands which extend radially outward to the periphery, often involving the pleura. In the development of this fibrosis there may result large or small bronchiectatic cavities and stenosis of the bronchi. Such a type is illustrated in figure 199. The patient was a young woman



FIGURE 199

Syphilitic fibrosis of right lung and pleura. Heart and trachea drawn over to right side. Edge of trachea and bronchus indicated by arrows.

who had outspoken symptoms of *tabes dorsalis*. She had no pulmonary symptoms and was unaware that she had any disease of the lung. There was a marked asymmetry of the chest. The whole right chest was dull to percussion, and, except in the upper lobe, the breathing was diminished. In the latter region there was tracheal (pseudoamphoric) breathing. The plate shows a marked retraction of the entire right chest. The heart and mediastinum are drawn over to the right. The extreme displacement of the trachea to the right apparently accounted for the amphoric breathing in the right upper lobe. There was in this case evidently a marked pleuro-pulmonary fibrosis. The absence of any pulmonary symptoms and tubercle bacilli in the sputum and the

strongly positive Wasserman reaction warrant a tentative diagnosis of syphilis. Of course, in this type of case, disappearance of the infiltration under antisppecific treatment is not to be expected.

These cases of syphilitic interstitial pneumonia, in spite of their great extent are remarkable for their benign character. One is struck by the almost entire absence of cough and expectoration, in which respect they are in marked contrast to fibroid tuberculosis, which they otherwise resemble. In their differentiation from tuberculosis and from other chronic non-tuberculous infections of the lungs, the positive Wasserman reaction is of transcendent value.

The cases of pulmonary syphilis which we have here presented illustrate the futility of any attempt to formulate, from the Roentgen standpoint, rigid criteria for diagnosis. Reflecting a varied pathology, the Roentgen plate exhibits a great diversity in the type of its shadows, none of which can be regarded as pathognomonic of this disease.

Clinically also there is little in the character of the symptoms to guide one to a correct diagnosis. In the presence of subacute or chronic inflammatory disease of the lungs, the clinician is bound to give precedence to other more common diseases, such as tuberculosis. Only the continued absence of tubercle bacilli, or some unusual feature of the case should direct serious attention to the possibility of syphilis. When this possibility is entertained however, great help may be derived from the Roentgen plate. In some cases at least, the appearance of the shadows will at once serve to exclude tuberculosis and the problem of diagnosis becomes narrowed to include suppurative lung disease, the various mycotic infectious and occasionally new-growths. It is at this stage of the problem that the Wasserman reaction may play a decisive role.

In conclusion, reference should be made to effusions in the pleura and other evidences of mediastinal pressure which may result from large gummatous masses at the root of the lung. In such cases the marked dyspnoea and the hemorrhagic character of the pleural effusion will naturally suggest a diagnosis of mediastinal neoplasm until the rapid disappearance of all the symptoms following antiluetic treatment.

CHAPTER XIII

Tumors of the Lungs

The tumors which arise in the thoracic cavity may be divided with respect to their most frequent origin into mediastinal and true pulmonary or pleural growths. Although anatomically these varieties are easily separable, it is not always possible to make the distinction between them from their clinical or even their Roentgen aspects. This is because pulmonary tumors usually spring from the root of the lung and therefore in the early stages of their growth appear to extend outward from the mediastinum very much as mediastinal tumors do. On the other hand the latter, in their further development often invade the lung and may produce both symptoms and Roentgen shadows similar to primary pulmonary neoplasms. Consequently, just as in other intra-thoracic diseases, the Roentgen diagnosis of tumors will fare best when it is firmly based on the clinical data. The age of the patient, the symptoms which marked the onset of the disease and the character of the secondary growths elsewhere in the body, will often furnish significant data which will throw light on the nature and anatomical situation of the primary tumor within the chest.

Pulmonary tumors may be conveniently classified for our purpose into primary and metastatic, because their Roentgen appearance serves to distinguish them from each other.

PRIMARY LUNG TUMORS

BENIGN TUMORS. As may be expected from their great rarity, benign tumors are not often encountered. From the Roentgen standpoint the essential characteristic of a benign tumor will be its failure to increase in size during a sufficient period of observation. Once a shadow suggestive of a neoplasm is discovered on the plate, we are bound to subject it to this test before the diagnosis of benign tumor may be seriously entertained. Even then, it will not be possible to exclude definitely inflammatory processes as a cause of the questionable shadow. A tentative diagnosis of non-malignant tumor of the lung was ventured in this spirit in the case illustrated in figure 200 after other possibilities were excluded.



FIGURE 200

A probably benign tumor of the lung.

The patient, a woman of twenty, suffered from an intractable form of asthma for one year. This was preceded by no circumstance which would suggest an inflammatory disease of the lung as its cause nor was evidence of sensitization to foreign protein obtained. Neither laboratory nor bronchoscopic examination threw any light on the origin of the condition. Although the Roentgen examination was frequently repeated during a period of eight months before she died, the circumscribed shadow illustrated above maintained its size and shape unaltered.

MALIGNANT TUMORS. Carcinoma. By far the largest number of primary lung tumors belong to the carcinomata. Their Roentgen appearance to a certain extent reflects the different pathological varieties of tumors and for this reason, with certain reservations, we may infer from the plate the particular type with which we have to deal. We can thus roughly recognize two forms, one beginning at the root of the lung and progressively involving more and more of its parenchyma

and a second which has a lobar distribution. The former is the adenocarcinoma which originates as an endobronchial tumor in one of the primary bronchi and spreads by extension through the wall of the bronchus to the adjacent lung. It is probable that dissemination along the peribronchial lymphatics also accounts for a considerable growth of the tumor. Lobar carcinoma on the other hand arises extrabronchially probably from alveolar epithelium anywhere in the lung; it is a tumor of flat cells which are often of indeterminate type.

LOBAR CARCINOMA

This term is applied in a descriptive sense to large tumors which involve a whole lobe in a dense neoplastic consolidation. Like most other primary cancers of the lung which we have observed, this tumor affects most frequently the upper lobes. Its origin outside the bronchial tree and the absence for a long time of any symptoms due to bronchial irritation explains its clinical latency. One is struck by the great size which these tumors have already attained when they begin to produce symptoms. On the plate the shadow of the tumor is often sharply



FIGURE 201

Medullary carcinoma of right upper lobe. Central Necrosis.

limited to one lobe, beyond whose confines it does not extend. Thus in figure 201 the tumor occupies the entire right upper lobe and is arrested sharply at the interlobar fissure. This was apparently a rapidly growing tumor, having a duration of only six months; during this time the major symptom was increasing dyspnoea, with only little cough. At operation, a necrotic medullary carcinoma was found. Because of their latency, it is only by a fortunate chance that such tumors are discovered early in their growth before they have involved an entire lobe. As will be seen in figure 202 such an early lobar carcinoma develops within the parenchyma of the lung, having no connection with the root and probably increases in size concentrically, unlike an adeno-carcinoma beginning in a bronchus.



FIGURE 202

Early stage of carcinoma beginning in the parenchyma of the lung.

The shadow of a lobar carcinoma in the left upper lobe will differ in shape from that in the right owing to the oblique direction of the inter-lobar fissure. Thus in figure 203 the lower border of the tumor has a curved contour which faithfully follows the line of the fissure. It will be observed that in carcinoma of the upper lobe, the extreme apex of the lung is frequently not invaded by the tumor.



FIGURE 203

Carcinoma of left upper lobe, lobar type. Phrenic paralysis. Lower border of tumor follows the line of the interlobar fissure.

BRONCHIAL CARCINOMA

The relative frequency of bronchial and lobar carcinoma is somewhat in dispute. This may partly be explained by the difficulty of formulating reliable statistics from a relatively small number of cases. From the Roentgen standpoint this difficulty is in part due to the various appearances of the tumors at different stages of their growth. Thus, a bronchial carcinoma arising from the root of the lung, may by a gradual

extension invade a whole lobe so that it will be indistinguishable from a true lobar neoplasm. Of the cases which we have observed, the bronchial, infiltrating tumor was the commoner type.

Although the bronchogenous tumors because of their situation produce symptoms much earlier than the lobar variety, nevertheless until they have extended through the bronchial wall into the lung, they cannot be directly demonstrated on the Roentgen plate. Their existence may occasionally be inferred from the fluoroscopic changes which result from stenosis of a main bronchus. Otherwise we are dependent almost entirely on the bronchoscopic examination for the diagnosis of these early cases.

Sooner or later these tumors grow through the wall of the bronchus and infiltrate the lung in the region of the hilum. The shadow of such a "hilum carcinoma" as it is called, cannot be distinguished in the beginning from the usual hilum shadows. For this reason, the Roentgen shadows in these early cases must be interpreted in the light of the symptoms from which they may acquire an unwonted significance. The following is a case in point:



FIGURE 204

Hilum Carcinoma. Adeno-carcinoma of right main bronchus beginning to infiltrate the lung. See figure 205.

A man, 65 years of age, otherwise well, began to have a constant unproductive cough, with paroxysms of dyspnoea. He occasionally raised some blood-tinged sputum. Because of his age and the dyspnoea a Roentgen examination was made, and the small, sharply defined triangular shadow seen in figure 204 was found at the root of the right lung. The unusual shape of this shadow, together with the symptoms warranted a strong suspicion of new-growth. This was verified by the excision of a small piece of the tumor through the bronchoscope. The subsequent rapid increase of this shadow and its unusual bilateral extension as an infiltrating adeno-carcinoma of the lung are indicated in the plate (fig. 205) made four months later.

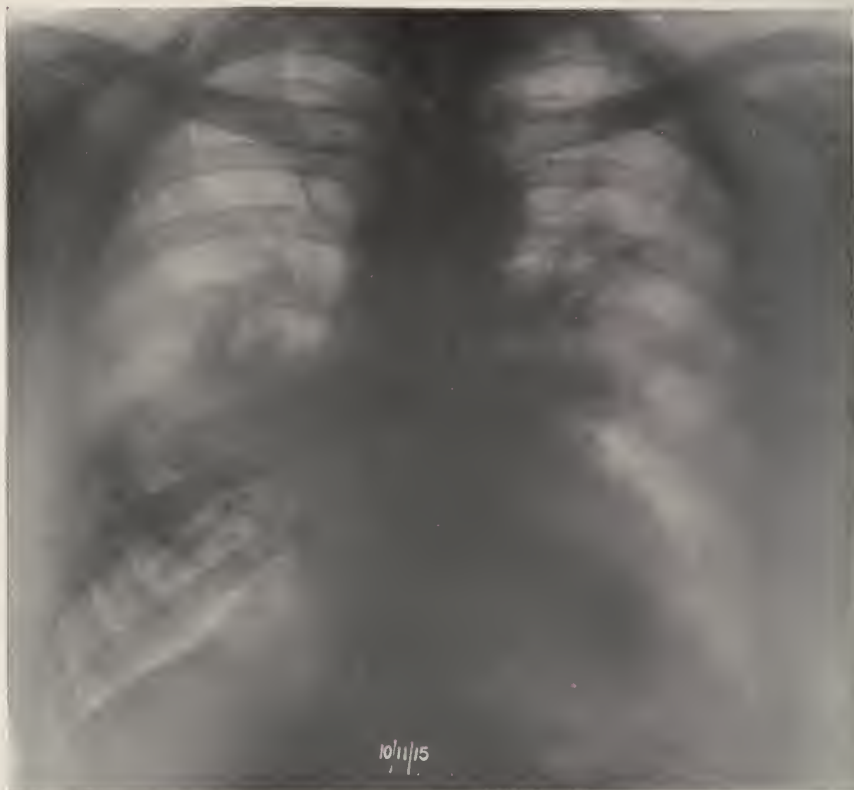


FIGURE 205

Bilateral adeno-carcinoma of the lung. Same case as figure 204 after an interval of four months.

The importance of re-examining patients with suspected new-growth for evidence of extension into the lung requires no further emphasis than is afforded by this case.

The larger bronchial carcinomata, when they involve the whole or a greater part of a lobe, resemble somewhat the true lobar type. (Fig. 206.) They usually lack, however, the sharp outlines of the latter, their shadows are less dense and homogeneous and they exhibit an infiltrating border. These features of bronchial carcinomata are



FIGURE 206

Adeno-carcinoma of right upper lobe.

brought out in the extensive upper lobe tumor shown in figure 207. In the smaller tumors the infiltrating margin is usually very evident and serves to distinguish them from mediastinal tumors, which in other respects they closely resemble. (Fig. 205.)



FIGURE 207

Adeno-carcinoma of right upper lobe, showing areas of softening. Metastases in left lung.

Bronchial carcinoma may occur in a very unusual form as a diffuse bronchial or peribronchial growth in which a whole lung may be infiltrated by radiating nodular strands extending from the root to the periphery. The Roentgen picture is similar to that of chronic pneumonitis and if the clinical picture, as may happen, is that of inflammatory disease of the lung, the possibility of new growth may not occur to the examiner. The problem presented by this type of case may be realized by the following history:



FIGURE 208

Carcinoma of lung resembling chronic inflammatory disease. Heart and mediastinum displaced to side of tumor.

A man of 47 years had cough and purulent expectoration for three years. There was occasional hemoptysis. For four months, in addition, he was very weak and had pain in the back and slight dyspnoea. He was much emaciated. Over most of the left chest there was dullness with distant bronchial breathing, excepting at the base where the respiratory murmur was diminished. This was later found to be due to a small hemorrhagic pleural effusion. Bronchoscopic examination disclosed a squamous-cell carcinoma which nearly filled the left main bronchus. The plate, (fig. 208,) sets forth this unusual type of tumor in which the cancerous deposits appear to follow the course of the bronchi to the periphery of the lung. The similarity to a chronic inflammatory process is maintained even to a displacement of the trachea and heart toward the diseased side.

SECONDARY CHANGES ASSOCIATED WITH NEW-GROWTHS OF THE LUNGS

When interpreting the shadows in the lungs which are presumed to be due to a tumor, we are often aided by pathological changes elsewhere in the chest. Thus, occasionally, either in the same or the opposite lung, small circular shadows of metastases may be seen which place the nature of the main shadow mass beyond doubt. However, the metastases occur only in the later stages of the disease, at a time when the characteristics of the primary tumor are fully developed. (Fig. 207.)

A careful inspection of the ribs and bony thorax in general, should never be neglected. The discovery of a pathological fracture or the typical cancerous erosion of a rib may shed a decisive light on an otherwise doubtful infiltration in the lung. (Fig. 228.)



FIGURE 209

Alveolar carcinoma of left upper lobe, of two years' duration. Necrosis of the tumor resulted in formation of a large cavity with clinical picture of lung abscess. Recurrent laryngeal paralysis.

Large tumors of the lung are frequently necrotic and at autopsy or operation irregular cavities are found in them. On the Roentgen plate the cavities are rarely seen as they are obscured by the dense tumor about them. Thus, in the case illustrated in figure 201 although there was no sign of a cavity on the plate, at operation there was found an irregular excavation of the tumor, the size of a lemon. It is particularly the massive lobar tumors which are apt to undergo extensive necrosis. The greater part of the growth may thus slough out leaving only a rim of tumor tissue at its periphery. The resulting cavity may then be converted into an abscess by secondary infection. Neither clinically nor with the Roentgen plate is it then easy to distinguish between a new-growth and a lung abscess especially as these cases are apt to be chronic in their course. The large cavity seen in figure 209 was due to the necrosis of an alveolar carcinoma of the left upper lobe. Clinically, the duration, which was over two years and the expectoration of foul sputum in large amounts rendered the presence of an abscess of the lung likely. However, one significant symptom, a recurrent laryngeal paralysis, which at autopsy was shown to be due to invasion of the nerve by tumor growth, lent greater probability to the diagnosis of tumor.

The shadow which we see on the plate in a case of carcinoma of the lung, does not always correspond in its extent to the size of the tumor. At times a considerable portion of the shadow is the result of secondary changes in the lung from occlusion of the bronchi. In this manner even small bronchial tumors may, by obstructing the lumen, lead to inflammation and suppuration of the affected lung. These secondary processes may completely mask the underlying tumor. The Roentgen examination will then reveal an infiltration of the lung, whose great extent gives an entirely erroneous idea of the size of the tumor. Often only the bronchoscopic examination will discover the small endobronchial growth which is at the bottom of the trouble. It will be clear that the complicated clinical and Roentgen picture will require the most careful judgment in its interpretation and that even with our best efforts we will at times fall into error.

Stenosis of a bronchus by a tumor will, in the absence of secondary infection, result in a simple atelectasis of the affected lobe. The physical signs over the airless lung will be added to those of the tumor itself and they will therefore create an impression of more extensive disease than actually exists. On the Roentgen plate also, the shadow of the atelectatic lung will merge with that of the tumor and an incorrect idea of the size of the latter will be given. Thus in figure 210 the shadow which obscures the entire left base was due mainly to an airless lower lobe, whose bronchus was occluded by a small tumor at the root of the lung.



FIGURE 210

Small adeno-carcinoma of left lower lobe. Most of the shadow is due to atelectasis of the lung resulting from stenosis of bronchus.

In their influence on the position of the heart and mediastinum pulmonary new-growths observe no invariable rule. The massive lobar tumors are more apt to displace these structures than are the infiltrating growths. Upper lobe carcinomata usually displace the trachea to the opposite side, whereas the heart, unless the tumor is in apposition to its borders, may retain its normal position. (Fig. 201.) When the heart is not displaced by a massive tumor, investigation may show that this is due to an atelectasis of the affected lobe caused by an occluding tumor in the main bronchus. Evidently this mechanism explains the failure of the heart to be displaced to the right in figure 210. In exceptional cases the heart is drawn over toward the side of the tumor. This was observed in the case of diffuse bronchial carcinoma already described (fig. 208) and it is not improbable that here also a decrease in the volume of the affected lung consequent on a closure of the main bronchus, was responsible for the displacement.

ROENTGEN-CLINICAL CORRELATIONS

During the early growth of lung tumors, especially of those at the hilum, the physical signs which later result from their extension to the surface are absent and the clinician has reason to be grateful for the help which the Roentgen examination affords him. In the case of the larger tumors, it is unfortunate that their symptoms and signs are not unlike those which characterize other, less serious pulmonary affections. In their clinical aspects they are frequently lacking in those features whose presence alone make the diagnosis more than a shrewd guess. It is noteworthy that pulmonary cancers are rarely associated with the cachexia of tumors found elsewhere in the body. It is not unusual therefore to observe patients who appear to be in excellent physical condition, yet in whom an enormous carcinoma of the lung is found. We have especially in mind an infiltrating form of carcinoma of a lower lobe which for a year or more may pursue a relatively benign course during which no suspicion of new-growth will be aroused. The clinical picture and physical signs in these patients are those of a sub-acute or chronic pneumonia at the base. As the onset of the disease is a so-called "cold," its insidious progression finds a ready explanation in the diagnosis of a persistent pneumonia.

**FIGURE 211**

Carcinoma of right lower lobe, with chronic course, simulating indurative pneumonia.

The patient whose plate is reproduced in figure 211, for one and one-half years had cough with blood-streaked sputum and signs of a basal infiltration. At one time, frequent fevers, sweats and a leukocytosis led to a futile operation for empyema. Only the development of a rib metastasis toward the end of his illness threw light on the true nature of the malady. From the Roentgen plate alone the differentiation from a pneumonic process could not be made.

In other cases, after a period of months during which the clinical picture of indurative pneumonia may be presented, the tumor may break down and, becoming secondarily infected, induce all the symptoms and signs of a lung abscess. This sequence of events marked the course of the case illustrated in figure 212. There is here seen an infiltration

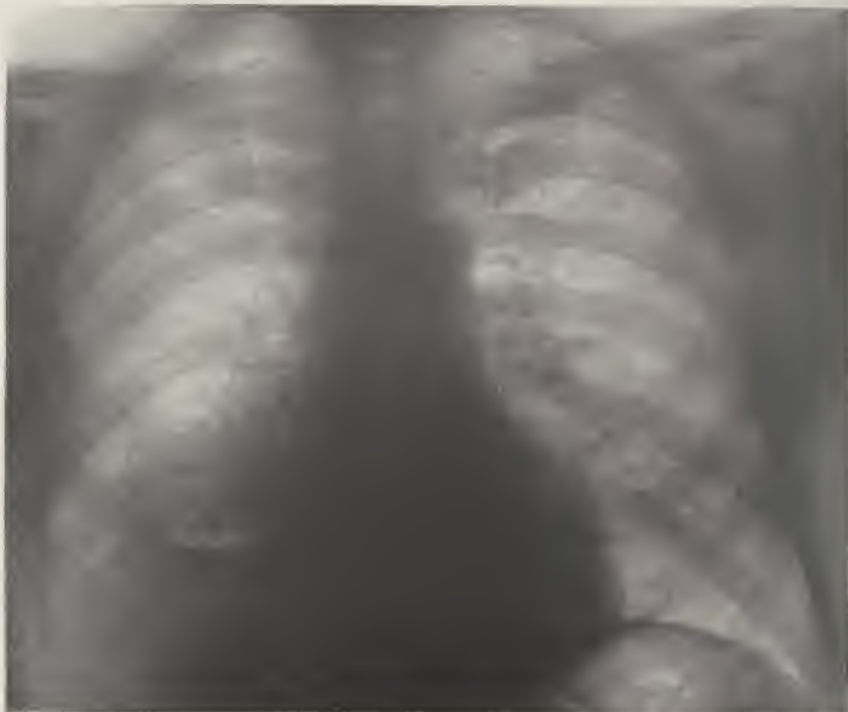


FIGURE 212

Carcinoma of right lower lobe. Slow breaking down of tumor with cavity formation. Clinical picture of lung abscess.

in the right lower lobe which is readily mistaken for a pneumonic process within which a cavity with a fluid level is seen. After a duration of six months frequent hemorrhages and expectoration of necrotic tissue prompted a bronchoscopic examination which revealed an adenocarcinoma of the right lower bronchus.

Such cases demonstrate the importance of keeping new-growths in mind as a possible cause of pulmonary symptoms which are first complained of at the cancer age.

The development of a pleural effusion secondary to a tumor of the lung introduces a number of new factors into the diagnostic problem which are of interest to both the clinician and Roentgenologist. It is usually a late phenomenon of the disease when the outspoken symptoms of the tumor have already manifested themselves. It may however arise during the early growth of the tumor and can then be explained as either due to an early involvement of the pleura or to the pressure of cancerous lymph nodes at the root of the lung. Such a pleural effusion, developing in an individual who previously has had few symptoms pointing to disease of the lung, may be the dominant feature in the clinical picture and may divert attention from the underlying tumor.

In the same way, on the Roentgen plate, the shadow of the fluid in the chest will obscure all evidence of disease of the lung. The significance of these "masking effusions," as they are called is well known clinically and their malignant origin is often at once evident from the red blood and tumor cells which they contain. After the pleural effusion has been evacuated it may be possible to see the tumor itself. The value of this procedure is emphasized by the following case:



FIGURE 213

Pleural effusion masking an underlying carcinoma of lung. See figure 214.

A middle-aged man complained of cough, expectoration and occasional dyspnoea for several months. The symptoms seemed to find a partial explanation in a fairly large pleural effusion in the right chest. (Fig. 213.) However, the occurrence of small hemoptyses aroused a suspicion of underlying lung disease, which was confirmed by a second Roentgen examination after most of the fluid was removed from the chest. The plate then showed a hilum carcinoma which had been effectually concealed by the pleural transudate, a small residue of which remained in the chest. (Fig. 214.)



FIGURE 214

Same patient as in figure 213 immediately after removal of pleural effusion. Shows hilum carcinoma. Small residual effusion at base and elevation of diaphragm. (arrow.)

Of the rarer pleural complications of lung tumors should be mentioned the empyemata which result from the rupture of bronchiectatic abscesses following stenosis of a bronchus by a new-growth. In these cases the Roentgen plate has only a limited value; the complicated picture in which the shadows of pleural effusion, inflammatory infiltration and tumor are commingled defy interpretation.

In recent years the bronchoscope, a new and valuable method has been enlisted in the diagnosis of pulmonary tumors. In the early diagnosis of bronchial carcinoma which, fortunately for the successful use of this instrument, commonly begins in the mucosa of a large

bronchus, the bronchoscope takes precedence over other methods of examination, in respect to both the accuracy and the timeliness of its findings. In the case of lobar carcinomata which presumably originate in the parenchyma of the lung, it is of less value, as the growth itself is not visible in the bronchial walls, except in the rare instances in which the latter are perforated by the extrabronchial tumor. Even in these cases, bronchoscopy often furnishes confirmatory evidence in the discovery of a narrowing, displacement or tortuosity of the bronchial canal which is caused by the pressure of the tumor mass. It is thus frequently possible by a correlation of the bronchoscopic and Roentgenologic findings to infer the pathologic type of tumor present and to bring it into harmony with the features of the clinical history. It will be found that the tumors which cast large massive shadows involving a whole lobe do not invade the bronchial tubes, although they may compress them. In these patients there is a long latent period during which there are few or no symptoms of bronchial irritation. On the other hand in those cases, whose Roentgen shadows correspond to the primary bronchial form of new growth, the bronchoscope almost invariably reveals an intra-bronchial carcinoma. In these patients, cough, dyspnoea and hemoptysis are apt to be early and prominent symptoms of the disease.

SARCOMA OF THE LUNG

By far the larger number of primary tumors of the lung belong to the carcinomata. Sarcoma is so uncommon that only some unusual feature of the case warrants its diagnosis. It is not unlikely that some of the cases of primary sarcoma which have been reported were in reality tumors which originated in the mediastinum and secondarily invaded the lung. The earlier incidence of sarcomata, the majority of which are found before the age of forty, may have some value in distinguishing them from epithelial tumors.

As far as we may draw any conclusions from the observation of a few cases, the Roentgen shadow of primary sarcoma is an extensive, dense, homogeneous one which has already reached an enormous size when the patient comes to the physician. This form of neoplasm is illustrated in figure 215. The entire left lower chest was occupied by a solid tumor which, on aspiration with a trocar was found to be a spindle-cell sarcoma. These tumors are extra-bronchial and they are therefore not apt to occasion much cough. The symptomatology of this case offered a striking contrast to that of a carcinoma of similar size, as it was marked by an absence of severe cough and dyspnoea and by the presence of high fever, sweats and rapid emaciation, which are uncommon even in advanced carcinoma. Both cases of primary sarcoma



FIGURE 215
Primary sarcoma of left lower lobe.

which we have observed occupied the lower lobes, a situation which is unusual in the cases of lobar carcinoma which we have nearly always found in the upper lobes.

In conclusion reference may be made to occasional cases in which the Roentgen plate shows a massive tumor of the lung whose size is out of all proportion to the mildness of the clinical symptoms. The absence of dyspnoea and cough and the continued good health of the patients for months or even years are so little consistent with malignancy that we are forced to regard them as relatively benign tumors, at least in the clinical sense. Two striking instances of this type of neoplasm are illustrated in figures 216 and 217. These patients remained in



FIGURE 216

Clinically benign type of lung tumor. Only symptom is slight dypnoea and dysphagia.



FIGURE 217

Clinically benign type of lung tumor. Practically no symptoms.

good health for many months with only minor thoracic symptoms, yet the physical signs, the history and the evidence of the exploring needle left do doubt of the existence of a solid tumor of the lung and in one of them, (fig. 218) perhaps also of the pleura. The pathology of these tumors remains unknown and we can only suggest that they are fibrosarcomata or endotheliomata or similar tumors of slight malignancy.

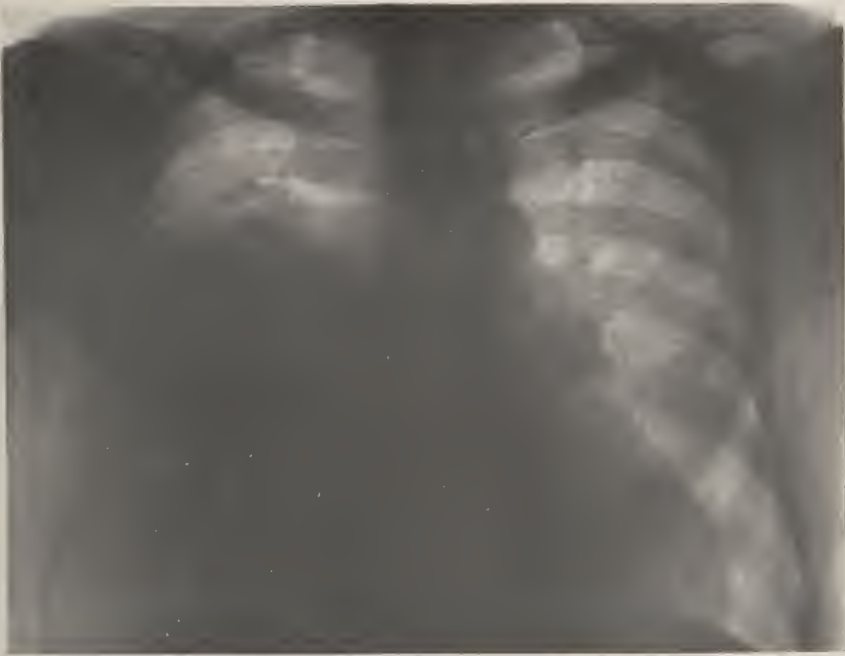


FIGURE 218

Clinically benign type of tumor of lung, probably involving pleura. Patient in good health for over one year.

DIFFERENTIAL DIAGNOSIS. From a study of the plates which have been presented to illustrate the Roentgen appearance of tumors of the lung, it will at once be evident that in certain cases a diagnosis can not be arrived at from these plates without reference to the clinical history. In some instances the shadows are little different from those occurring in such other pulmonary diseases as lung abscess, pneumonia and occasionally caseous tuberculosis of a lobe of the lung. We believe it wiser to recognize this fact and frankly to appeal to the history and physical signs for confirmation than to advance specious points of differentiation which are at best uncertain. A decade ago, the diagnosis of tumor of the lung was regarded as a brilliant accomplishment and a clinical tour de force; to-day it may truthfully be said that by a proper combination of clinical and Roentgen evidence, few pulmonary neoplasms need escape discovery.

CHAPTER XIV

Metastatic Tumors of the Lungs

Primary tumors in various organs so frequently occasion metastases in the lungs, that the Roentgen examination of the latter is endowed with an unusual importance. The clinician is thereby enabled, not only to discover metastases which elude his physical examination but he is also afforded decisive, though indirect evidence as to the nature of disease elsewhere in the body. This is fortunately facilitated by the distinctive character of the shadows which permits of their easy and early recognition.

In respect to their pathology, we have to do practically with three kinds of metastatic tumors namely, carcinoma, sarcoma and hypernephroma, differences in whose size, number and distribution in the lungs lead to a great diversity of Roentgen appearances. With rare exceptions, the metastases are multiple. The obvious reason for this is that secondary tumors are usually symptomless during their early growth and they are therefore not discovered until they have developed in great numbers.

The earliest metastases appear as small, faint, circular shadows which are clearly outlined in the lung. They are first seen in the lower lobes where they are apt to attain their greatest size. As they increase in size they usually retain their circular shape, although occasionally, especially in the case of carcinomata and hypernephromata, they may become oval or irregular in outline. Sarcomata on the other hand, even when they occupy a whole lobe, usually remain smooth and circular.

As a rule it is not possible to make a pathological distinction between the various forms of secondary tumors. In general it may be stated that the very large tumors with sharply circular outlines are sarcomatous. The carcinomata are of slower growth and for this reason the patients succumb to the disease before the secondary tumors have attained a large size.

SARCOMA. Metastatic sarcoma of the lung is in a large proportion of the cases secondary to tumors of the bones. In one form the tumors are few in number and they increase in size with astounding rapidity. This feature is brought out in figure 219 in which the greater part of the left upper and lower lobes was already involved four weeks after amputation of a leg for periosteal sarcoma.



FIGURE 219

Large metastases in left upper and lower lobes and at root of right lung. Primary tumor periosteal sarcoma.

In other cases the individual neoplasms are smaller and more numerous. The tumors shown in figure 220 were discovered six months after amputation of a lower extremity for a fascial sarcoma. While this type of tumor usually presents no difficulty in diagnosis, it is not always easy to distinguish an extensive metastatic involvement of the



FIGURE 220

Multiple small metastases, secondary to fascial sarcoma of thigh.

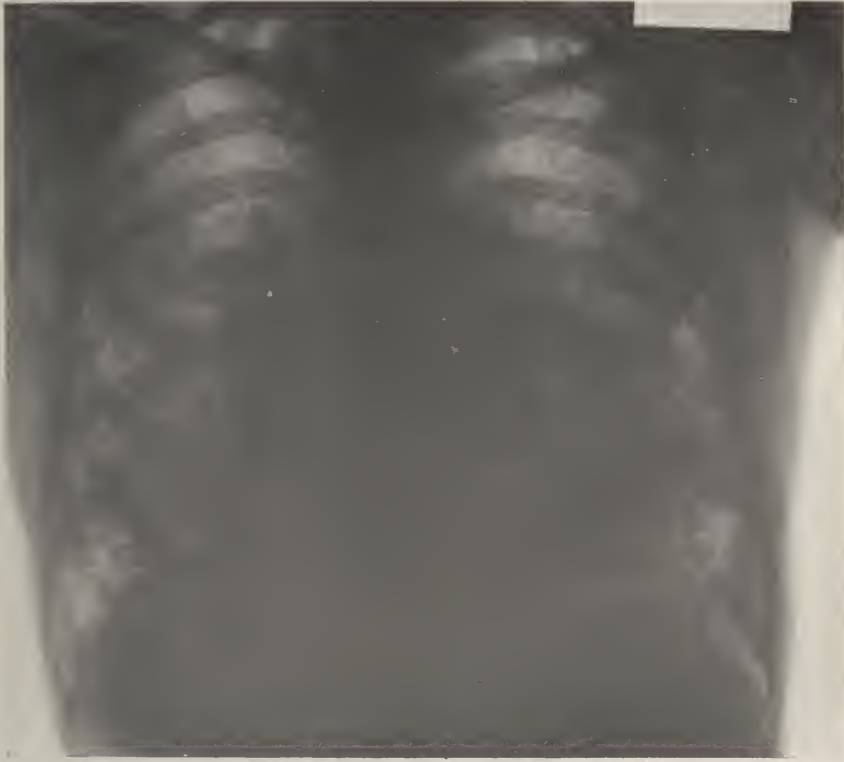


FIGURE 221

Metastatic sarcoma. Primary tumor in femur.

lung from certain forms of pulmonary tuberculosis. Thus in figure 221 the individual metastases are so small and numerous as to resemble a confluent, lobular tuberculosis. We are aided in the distinction by an absence of disease at the apices and by finding somewhere in the lower lobes typical circular metastatic tumors.

An unusual form of metastatic sarcoma, secondary to a tumor of the testis is shown in figure 222.



FIGURE 222

Metastases secondary to sarcoma of testis. Note irregular shape and lobulation of tumors.

CARCINOMA. We are here confronted with a greater diversity of shadows which depends on the numerous sources of the primary growth. The most common Roentgen type is illustrated in figure 223 in which the tumors were secondary to a primary neoplasm in the gastro-intestinal tract.



FIGURE 223

Metastatic carcinoma secondary to carcinoma of the stomach.

It is characteristic that the tumors increase in size and number from the apex to the base, although exceptions are found, as in the occasional case of large metastases in the apex. (Fig. 224.)



FIGURE 224
Metastatic Carcinoma.

More irregular tumors, both as to shape and distribution, may occur secondary to thyroid and prostatic new-growths. Thus in figures 225 and 226 the neoplasms are marked by an irregularity in shape and also in density not often found in the other forms.



FIGURE 225

Metastatic carcinoma. Primary in thyroid. Note shadow of thyroid gland at root of neck.



FIGURE 226

Metastatic carcinoma secondary to tumor of prostate.

Secondary carcinoma of the lung from primary testicular tumors appears to be exceptional in its development and resembles in all respects the larger sarcomatous metastases. In figure 227 is pictured a large tumor of the right lung which was discovered two months after the removal of a carcinomatous testis. The effusion in the left pleural cavity speaks for an involvement of that side also. It is remarkable that with such an extensive growth, the patient was ambulant and had but minor symptoms consisting of cough with occasional hemoptysis for a period of four weeks.



FIGURE 227

Large metastasis involving right lower lobe secondary to carcinoma of the testis. Left pleural effusion.

A rare variety of metastatic carcinoma known variously as miliary carcinosis or carcinomatous lymphangitis of the lung is worthy of mention, both because of its unique pathology and its remarkable Roentgen shadows. The lungs are studded with innumerable miliary and submiliary nodules which apparently are disseminated by way of the lymphatics both within the lung and on the surface of the pleura. On cursory inspection the plate closely resembles a miliary or submiliary tuberculosis, yet on a more careful examination, especially in the less densely infiltrated portions of the lungs, the shadows are seen to have a smoothness of contour and a circular shape not found in tuberculous lesions.

In the literature miliary carcinosis of the lungs has commonly been associated with primary tumors in the gastro-intestinal tract. We have found them also secondary to carcinoma of the breast and of the lungs and pleura and they have been remarkable for the long latent period which has intervened between the onset of the primary tumor and the development of the metastases. In figure 228 is illustrated a well

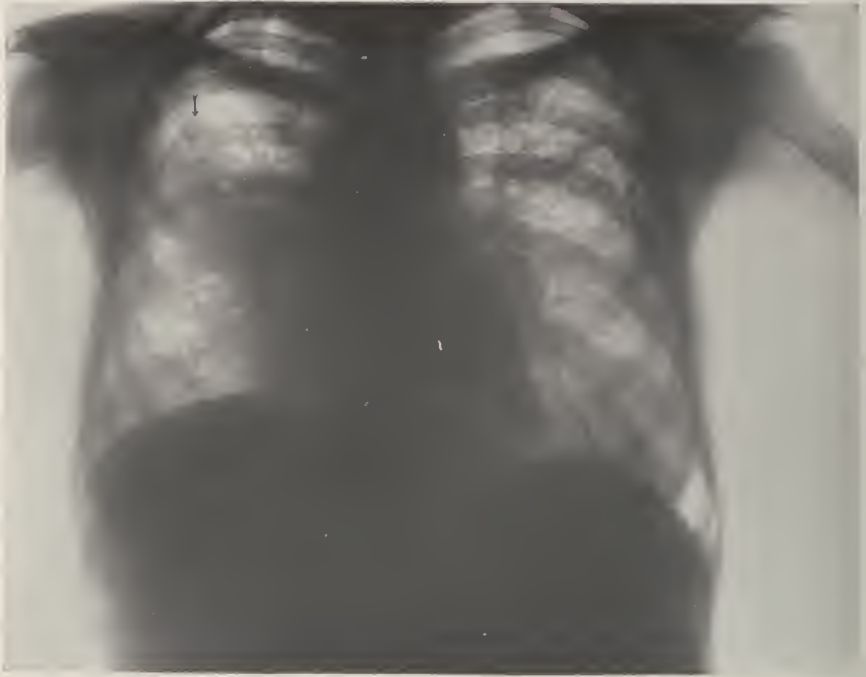


FIGURE 228

Miliary carcinosis of left lung. Primary adeno-carcinoma at root of right lung. Arrow indicates metastatic growth and erosion of rib.

developed example of this disease which was secondary to an adenocarcinoma of a bronchus. In our experience these cases have usually been regarded clinically as tuberculous, an error to which the Roentgen plate if casually studied, may contribute. However, the absence of fever, the disproportion between the extent of the lung involvement and the mildness of the symptoms and finally, evidence of metastases elsewhere should remove all doubt as to the nature of the disease.

HYPERNEPHROMATA, as a rule, bear resemblance to the carcinomatous tumors. They occur either as numerous small growths or as larger ones in lesser numbers which occasionally exhibit an infiltrating border. The metastatic tumors in figure 229 were found in a patient two months after the removal of a hypernephroma.



FIGURE 229

Metastatic Hypernephroma.

The complications of metastatic growths which may be observed on the plate are few. Pleural effusions are associated usually with the larger tumors. A cortical neoplasm may rupture into the pleural cavity and produce either a free or encapsulated sero-pneumothorax. (Fig. 230.)

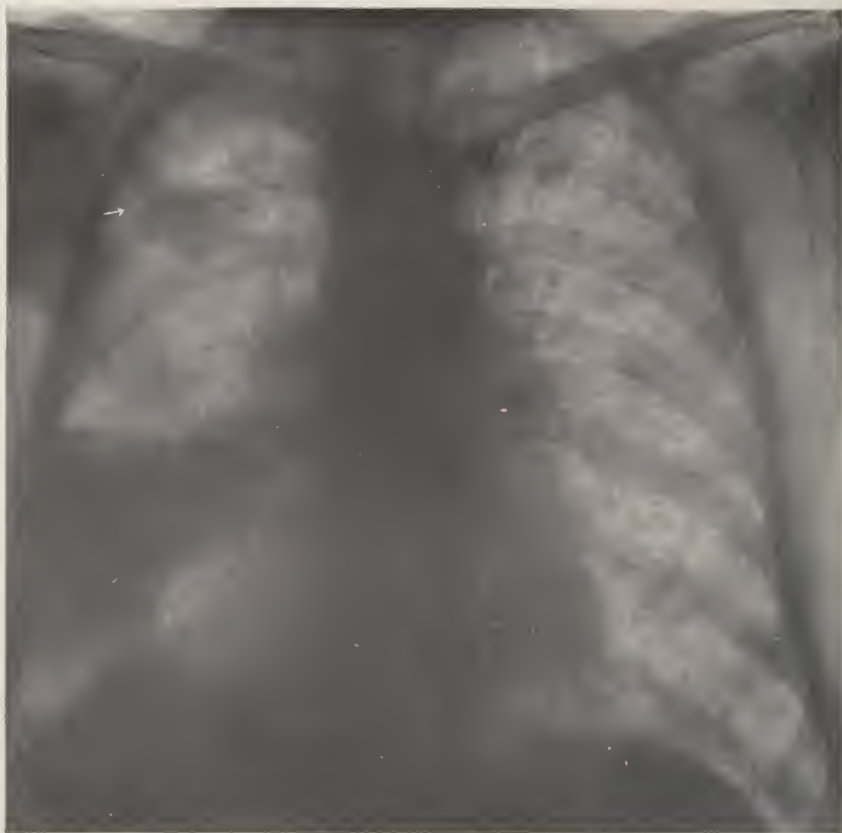


FIGURE 230

Miliary metastases in left lung secondary to carcinoma of bronchus. Perforation of cortical tumors with localized sero-pneumothorax. Note two fluid levels in right chest.

ROENTGEN-CLINICAL CORRELATIONS

It may not be amiss to dwell on the practical importance of the Roentgen examination of the chest for evidence of secondary new-growths. To the surgeon, who is debating the excision of a primary tumor or the amputation of a limb, it is a question of superlative importance whether there is a secondary involvement of the lungs. It is evident that if metastases have already developed, he may desist from operation altogether; in the case of a tumor of an extremity, a cure being precluded, he may be content to excise the new-growth rather than to subject the patient to a fruitless amputation. It has been our experience on more than one occasion to find metastases in the lungs shortly after operative removal of the primary tumor; these metastases were undoubtedly present previous to operation and their discovery

would certainly have influenced the surgeon against a radical interference. The tendency of bone sarcomata and hypernephromata to metastasize very early in the lungs should make a pre-operative Roentgen examination of the lungs a routine procedure.

Metastatic tumors in their early growth are absolutely latent both as to symptoms and physical signs and extensive metastases in the lungs are consistent with apparently robust health and good color. Even when they have attained large proportions they may produce neither subjective symptoms nor dulness nor altered breathing. For example, the large tumor in the right lower lobe in figure 227 was unrecognized clinically, because it was centrally situated and was covered by normal lung.



FIGURE 231

Metastatic tumors secondary to carcinoma of breast. Destruction of 5th dorsal vertebra by secondary tumor.

While the positive Roentgen finding of metastases has thus great clinical value, it is unfortunate that a negative plate does not absolutely exclude them. Thus, the extensive infiltration of the lung in figure 221 was discovered shortly after amputation of the thigh for sarcoma, although the lungs immediately before operation showed no abnormality. It is probable that at the time of the first Roentgen examination the tumors were in process of growth and were still too small to be demonstrated. The possibility exists, of course, that the malignant cells were mobilized by the manipulation during operation. It is particularly in cases of carcinoma of the breast that it is difficult to exclude metastatic growths. The cancer cells may apparently lie dormant in the bronchial nodes for some time and months or years may elapse before metastases are found on the plate. The extensive involvement of the lung in figure 231 was thus found over a year after removal of the breast.

Equally with the surgeon, the internist has much to gain from the Roentgen examination in cases of suspected metastases. In patients with obscure disease elsewhere in the body, the lungs offer a fertile and accessible field for investigation in which at times the most surprising disclosures come to light. Of such diseases we may instance severe



FIGURE 232

Large metastasis from carcinoma of stomach.

anemia for which no cause is found, but in which the suspicion ever arises that an undiscovered neoplasm, perhaps in the stomach, is at the bottom of the trouble. A latent carcinoma of the gastro-intestinal tract may thus incite a profound anemia which will challenge the acumen of the examiner until its cause is betrayed by the discovery of a lung metastasis. The large metastasis of the left upper lobe in figure 232 was thus found in the course of a systematic examination of the viscera for evidence of a new growth in a patient who for several months presented a progressive anemia of the aplastic type. The discovery of a metastasis directed attention at once to the stomach, where a carcinoma, which had produced no direct symptoms, was found. There come to mind also cases in which the only sign of pulmonary disease consists of repeated hemoptyses which are apt to be attributed to tuberculosis of the lungs. In the case illustrated in figure 229 it was only the discovery of pulmonary metastases which prompted an investigation of the urinary tract, where a large hypernephroma was found. It is particularly in the cases of miliary carcinoma of the lung that this confusion with pulmonary tuberculosis is apt to occur. On more than one occasion we have found, in patients whose only symptom was hemoptysis, a universal involvement of the lung in a miliary cancer. Because of an almost entire absence of physical signs these were regarded as cases of incipient pulmonary tuberculosis. (Fig. 230.)

SECTION V

The Pleura

Dry Pleurisy

Pleural Effusions

Pneumothorax

Tuberculosis of the Pleura

Tumors of the Pleura

Chronic Pleurisy

CHAPTER XV

The Pleura

There are few intrathoracic diseases, in which the Roentgen examination is attended with more interesting and instructive disclosures, than in those which involve the pleural membranes. The great extent of the pleura and its proximity to important organs both in the chest and abdomen make it a frequent site of pathologic processes of the most varied sort, to the diagnosis of which the Roentgen Ray is peculiarly adapted. Owing to the intricate topography of the pleural membranes, however, the Roentgen interpretation encounters more than the usual difficulties, especially in respect to pleural effusions. Whether the latter are situated on the anterior or posterior aspect of the chest or buried within the depths of a fissure or confined in the mediastinal recess, they lose their topographical features when they are projected on the plate and become merely flat shadows. Our interpretations then have imposed upon them the task of reconstructing from simple shadow masses, the spatial relations of effusions which are frequently complicated. In spite of these inherent difficulties, the Roentgen diagnosis of pleural diseases has achieved a remarkable exactitude which has relieved the physical examination of much of its uncertainty. No longer need indecision attend the distinction between such clinically similar conditions as a small pleural effusion or congested bases nor need one for days remain in doubt regarding the development of empyema following a pneumonia, before definite physical signs indicate its presence. These and numerous other questions which are constantly arising during the clinical course of a case are now quickly and decisively resolved by the Roentgen examination.

DRY PLEURISY

The Roentgen diagnosis of dry pleurisy is apt to be as disappointing as the search for pleuritic rales. The cause of the thoracic pain so often complained of in rheumatism or latent tuberculosis is only rarely evident on the plate, as a thin fibrinous deposit on the pleura is completely permeable to the Roentgen Ray. Only in situations where the pleura is reflected into a fissure will such an exudate be visible. It most often takes the form of a sharp linear shadow at the site of the right upper fissure which extends transversely across the chest. (Fig. 235). This shadow may also be found in cases of chronic pulmonary disease and therefore it only has a significance in the diagnosis of acute pleurisy if the symptoms indicate that the process is a recent one. Larger fibrinous masses, with corresponding Roentgen shadows, appear

to result from pyogenic infections and are more apt to be seen in the fibrino-plastic pleurisy secondary to lung suppuration or subphrenic abscess. The character of these shadows is shown in figure 381.

Acute inflammation of the pleura is usually accompanied by an inhibition of the movement of the diaphragm. In patients who have pleuritic pain the discovery of an elevated, immobile diaphragm on the affected side, even in the absence of shadows of a pleural exudate, will be strongly suggestive of a pleurisy. This phenomenon is most marked on the left side where the diaphragm, unsupported by the liver, readily yields to the upward pressure of the intestines.

PLEURAL EFFUSIONS

Fluid may accumulate freely in the pleural cavity or it may be localized by adhesions or confined in one of the many pleural recesses. We shall first discuss free effusions as they are the more usual form.

FREE EFFUSIONS

The Roentgen features of pleural effusions are determined first by the great absorptive power of fluid for the Roentgen Ray, which causes it to cast a dense homogeneous shadow completely obscuring the lung beneath it and secondly, by the elasticity of the lung which is responsible for the unique configuration of the shadow. We may best observe these features in non-purulent effusions such as the hydrothorax of cardiac disease. The increase in the size of the effusion is here often so slow that the various stages of its accumulation may be minutely studied. The first evidence of fluid will always be found at the base, in the costo-phrenic sinus, which becomes obscured by a dense shadow. The upper border of this shadow is concave. (Fig. 233.) As the effusion increases, the shadow extends progressively upward, being considerably higher in the axilla and sloping obliquely down toward the diaphragm. (Fig. 234.) The upper border of the shadow is sharply defined from the lung as long as the effusion is still small. However, as it enlarges, owing to a compression of the lung, the latter becomes less aerated and the distinction between fluid and lung is less clear. In some cases, especially with purulent effusions, fibrinous deposits on the surface of the lung will have the same effect. For these reasons, a pleural effusion may actually not be as large as its shadow would lead one to believe because the compressed lung contributes materially to the extent of the shadow. A pleural effusion is usually, though not always, to be distinguished from a consolidation of the lung, by the greater density of its shadow which



FIGURE 233

Small effusion at right base with oblique level.



FIGURE 234

Moderate sized pleural effusion. Tuberculous infiltration, left infraclavicular region.

approximates that of the liver below it. Its shadow is therefore continuous with that of the liver whose upper border it early obscures so that the diaphragm is not visible.

The Roentgen plate has amply confirmed the deductions of Garland in regard to the configuration of the area of dulness in pleural effusions. The upper surface of fluid owes its shape to a combination of antagonistic forces, namely, the weight of the fluid and the elastic tension of the lung. The former causes the effusion to gravitate to the base of the chest; the latter, as the lung is separated from the chest wall, exerts a pull on the surface of the fluid, which is greatest near the lower axilla, drawing the fluid upward. The effusion therefore rises obliquely from the mid-line, reaching its highest point in the axilla, thence descending toward the spine, once more obliquely. This so-called "S" curve will, in different individuals, vary in height and

obliquity depending on the elasticity of the lungs. Thus, in young persons, the elasticity of whose lungs is unimpaired, the margin of the shadow takes a more perpendicular course, reaching a higher point in the axilla.

The movement of free fluid in the chest may be demonstrated both on the plate and on the fluoroscopic screen. When the patient is recumbent the fluid flows upward to the apex so that the pulmonary field is entirely obscured and the level of the fluid disappears. On the screen, the movement of the fluid level may be seen as the patient's chest is tilted to the side. Unless there is air in the pleural cavity, this shift in the fluid level is a slow, almost imperceptible one. It is best seen with effusions of moderate size. When the chest is almost full of fluid, or in the presence of adhesions on the pleura, it may be absent.

PURULENT EFFUSIONS. The Roentgen features which we have thus far considered apply particularly to nonpurulent effusions. In empyemata, on the other hand, the early inflammatory exudate may not be fluid and for this reason the Roentgen appearance, at least in the beginning, will differ from that which we have found in transu-



FIGURE 235

Beginning empyema with plastic exudate over right lung. Note retraction of lung from chest wall with narrow shadow of exudate. Right lung hazy and interlobar fissure outlined by pleural thickening.

dates. As is well known, in the early stage of an empyema, the pleura may be covered by a shaggy fibrino-purulent exudate of some thickness, within whose interstices a small amount of fluid may be enclosed. Such a layer of fibrinous exudate completely encasing the lung may be recognized on the plate by a distinctive shadow, which is visible in the axilla as a narrow band extending from apex to the base. This shadow is due to a slight retraction of the lung, between whose surface and the chest wall the exudate is interposed. The peculiar shape and situation of this shadow is due to the incidence of the ray as it tangentially traverses the edge of the lung and the exudate. The influence of the exudate on the surface of the lung will be seen as a general decrease in the illumination of the pulmonary field. The following illustrations depict the Roentgen features of these early empyemata. In figure 235 is seen a beginning empyema in a child recovering from pneumonia. It will be noted in the first place that the entire right lung is poorly aerated. The lung is also retracted from the chest wall, from which it is separated by a narrow shadow of uniform width, due to a non-fluid



FIGURE 236

Beginning empyema after pneumonia of right lower lobe. Very thick fibrino-plastic exudate over lung.

pleural exudate. The width of this shadow varies in different cases, depending on the thickness of the inflammatory deposit. Thus in figure 236 the axillary shadow is of considerable width.

This shadow of a fibrino-purulent exudate on the pleura is of the greatest value in the early diagnosis of empyema complicating pneumonia, as it precedes definite physical signs of an effusion and it is found before pus can be obtained in any quantity by the exploring needle. Thus in figure 237 and 238, although the pneumonic process in the right lower lobe is still at its height the evidence of a beginning pleurisy is unmistakable. A narrow axillary shadow extends from the apex to the base and it can even be recognized on the surface of the consolidated lower lobe.

The early fibrinous deposit on the pleura is followed usually in a few days by a true fluid exudate which gravitates to the base of the chest. When the pleural effusion is secondary to pneumonia of the



FIGURE 237

Pneumonia of right lower lobe with beginning empyema. Lung separated from chest wall by fibrino-purulent exudate. See figure 238.

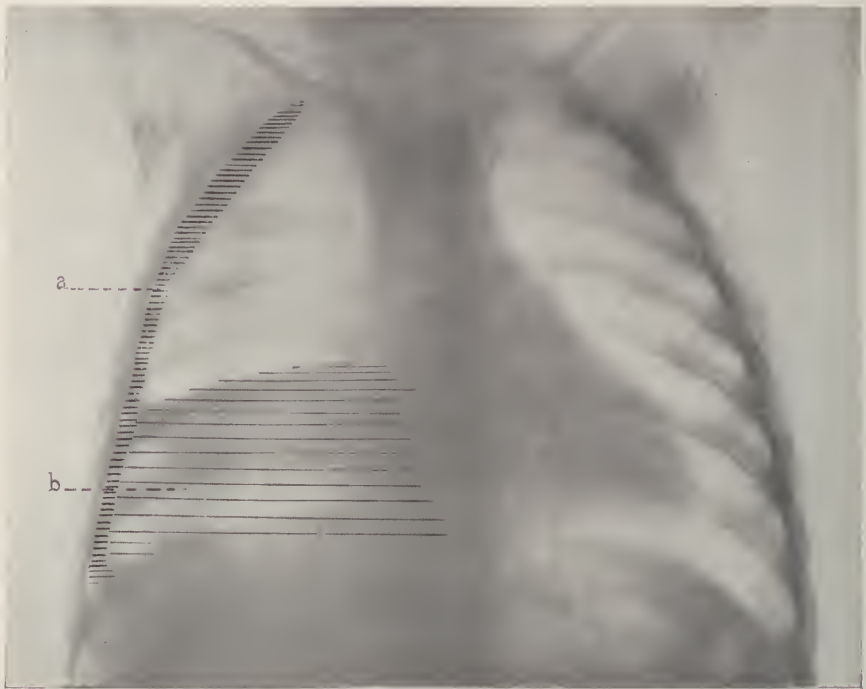


FIGURE 238

Diagrammatic representation of figure 237.

a.....Fibrino-purulent pleural exudate.

b.....Pneumonia of right lower lobe.

lower lobe the shadow of the former is accordingly superposed on that of the latter. It is, however, so much denser than the pneumonic shadow that the demarcation between the lung above and the liver below is lost. The consequent disappearance of the curve of the diaphragm may therefore be looked upon as the characteristic sign of a purulent effusion, complicating lower lobe pneumonias. Thus in figure 236, the upper half of the chest exhibits a fibrinous nonfluid exudate, whereas at the base there is a composite shadow of pneumonia and fluid.

ENCAPSULATED EFFUSIONS

Although free pleural effusions offer few difficulties in diagnosis which cannot be overcome by the experienced examiner, this is not true of the encapsulated variety. The physical examiner is at a great disadvantage when he seeks to determine the size, shape and location of the almost endless variety of atypical effusions which may form within the pleural cavity. The Roentgen Ray on the other hand labors under no

such disadvantage. The limits of the effusions are unerringly defined and collections of fluid deep within the chest are pictured with the same precision as those more superficially situated.

Pleural effusions are more often encapsulated than is commonly believed. It is not unusual for even very large effusions to be walled off by adhesions and although the latter are not discoverable by physical examination, they are usually evident on the Roentgen plate. There are two major influences which determine encapsulation of a pleural effusion, first, the character of the exudate and secondly the extent of the respiratory movement of the chest wall. Undeniably, infection plays a predominant role in determining the sacculation of an effusion. Evidently this is because the plastic exudate of an inflammatory process tends to agglutinate opposing pleural surfaces and thus to wall off the effusion. For this reason, empyemata are frequently encapsulated whereas the transudates of cardiac or renal disease or of neoplasms are rarely so. In our discussion of encapsulated effusions, we are therefore concerned mainly with those of inflammatory origin, be they acute, purulent and pyogenic or chronic, non-purulent and tuberculous.

The second factor involved in encapsulation is of lesser importance and determines the situation of the limiting adhesions. The mobility of the chest during respiration is not everywhere of equal extent. It is greatest in the axillary and basal regions, whereas it is much restricted at the apex and toward the mediastinum. In the latter situations, the movement of the pleural membranes is accordingly less active and adhesions can more readily form. We therefore find encapsulated empyemata oftenest in the axillary half of the chest and rarely, at least primarily, in its mesial portion. Finally, the reflex rigidity of the chest wall which results from an inflammation of the pleura will also contribute to a limitation of the respiratory movement and will thus facilitate the formation of adhesions.

The Roentgen shadows of encapsulated effusions exhibit the greatest variety in respect to their size, shape and situation, this being dependent mainly on the location of the limiting adhesions. For our purposes we may most conveniently divide them as follows:

A. SUPERFICIAL EFFUSIONS

These are effusions encapsulated in the general pleural cavity or "parietal encapsulated effusions."

B. CONCEALED EFFUSIONS

I. Effusions encapsulated between the lung and the diaphragm. "Diaphragmatic or Infra-pulmonary effusions."

II. Effusions encapsulated between the lung and the mediastinum. "Mediastinal Effusions."

III. Interlobar Effusions.

This classification, which emphasizes only the topographic features of these effusions is of practical importance, because it lays the basis for an exact localization of the fluid to the end that exploratory aspiration and subsequent operation may be performed with the greatest precision. This, after all, is the main purpose of the Roentgen examination.

(A) SUPERFICIAL OR PARIETAL ENCAPSULATED EFFUSIONS. These represent the commonest variety of sacculated effusions. They are usually secondary to pneumonia or cortical abscesses of the lungs; in rare instances they are metastatic infections from foci elsewhere in the body. As a result of the fibrinous adhesions which wall off the fluid at one or more points, the lung cannot retract from the chest wall and consequently its elastic tension is not exerted on the effusion in the usual manner. We will accordingly note in most cases an absence of the oblique upper surface of the fluid, which is so characteristic of free effusions. One of the simplest forms of encapsulation is illustrated in figures 239 and 240. In this case an adhesion in the region of the interlobar fissure has divided the pleural cavity into an upper and a lower half, in each of which the usual intrapleural forces have come

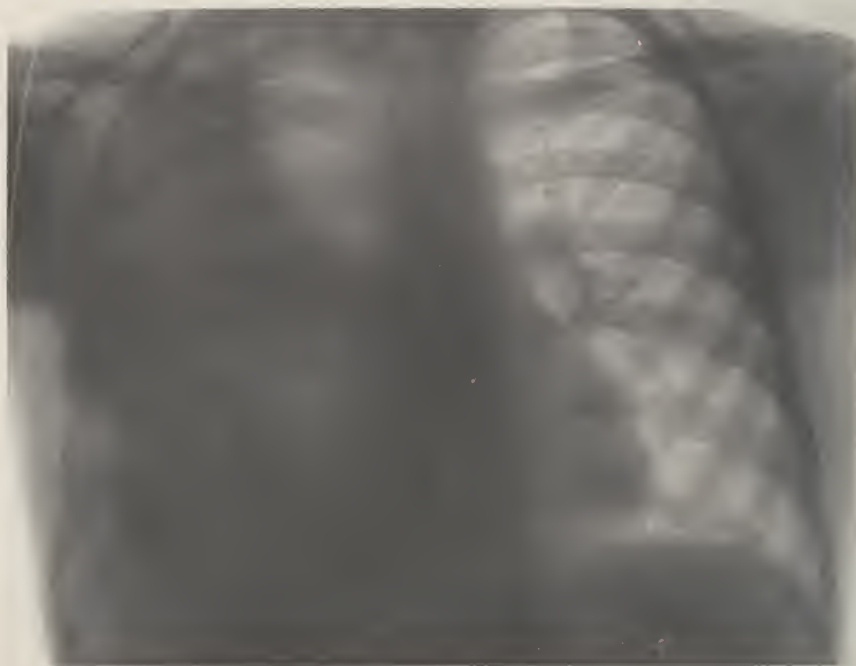


FIGURE 239

Pleural effusion divided into two sacculations by adhesions at the interlobar fissure. See figure 240.

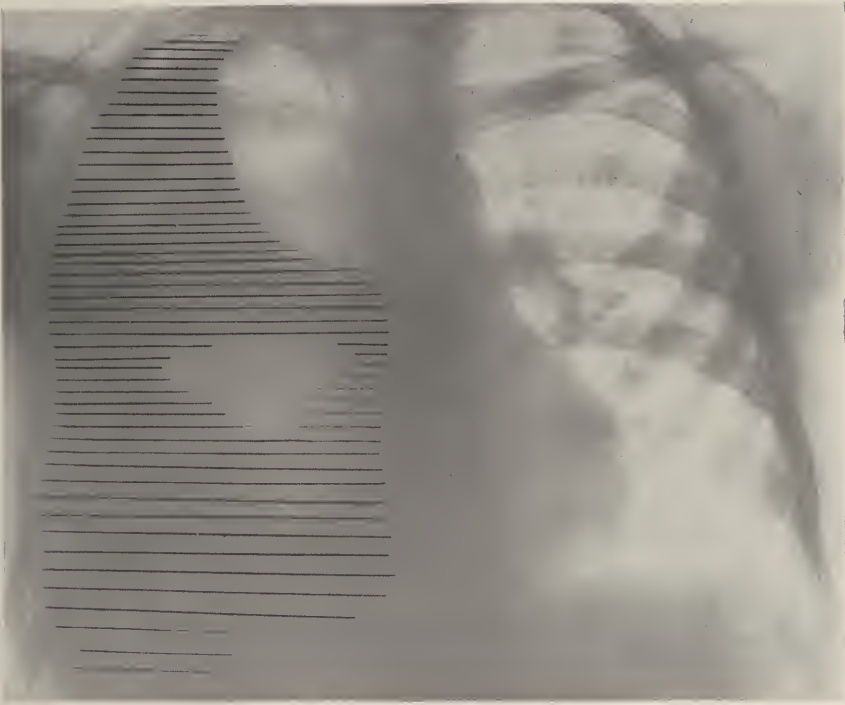


FIGURE 240

Diagram of Figure 239 showing outlines of two separate effusions.

into play, in such a way as to reproduce in miniature two "S" curves. Evidently the upper and lower lobes have separately retracted and drawn the fluid upward with them.

The fluid may occupy any part of the general pleural cavity and we may accordingly recognize an axillary, a basal, an apical and a mesial form of encapsulated effusion.

(1) AXILLARY EFFUSIONS. The most frequent site for the encapsulation of pus is in the axillary portion of the chest, the adhesions being situated either in the anterior or posterior axillary region, or both. The character of the Roentgen shadows in these cases is illustrated in figure 241, where the empyema occupies the lateral half of the chest



FIGURE 241

Effusion encapsulated in lateral half of chest.

from apex to base. The adhesions do not always limit the empyema strictly to the axillary region. It may encroach on the apex or on the base and thus produce a greater variety of shadows. In figure 242, for example, the axillary effusion is large and also invades the apex of the chest. As is so often the case with purulent pleurisy, this one consisted of two sacculations which were completely separated from each other and required two operations for its evacuation.

The clinical features of axillary empyemas may profitably detain us for a moment because of the light which may be thrown on them by the Roentgen examination. The physical signs are often puzzling especially in respect to percussion dulness. The extreme dulness of free effusions is often lacking, owing to the fixation of the lung along the edge of the empyema. It is especially noteworthy that in the presence of an axillary effusion which extends to the posterior aspect of the lung, a loud tympanitic note may be heard anteriorly. The diagnosis may be further confused by the occurrence of numerous rales along the borders of the effusion.



FIGURE 242

Bilocular encapsulated empyema.

It is, however, particularly in respect of exact localization and exploratory aspiration that a consideration of these unusual signs is important. In aspirating a free effusion, the operator is accustomed to insert the needle in the scapular line at the base of the chest, where fluid is usually obtained without difficulty. In axillary effusions, on the other hand, aspiration at this point is often unsuccessful because the fluid is more laterally situated. Dulness, if present at this site may be due to thickened pleura at the margin of the empyema or to compressed lung, rather than to the effusion itself. Therefore whenever there is reason to suspect an encapsulation of the empyema, it is wise to aspirate either in the anterior or posterior axillary line. With a single puncture the needle may be pointed in any direction and usually the pus will be discovered. Needless to say, when aspiration is performed on the left side in the anterior axillary line, one must inform oneself by a previous Roentgen examination of the exact position of the heart, lest it be injured.

(2) BASAL EFFUSIONS. An empyema at the base of the chest is not uncommonly found by the surgeon to be encapsulated, although this may not have been suspected from the physical examination. Similarly the Roentgen plate may fail to reveal the presence of adhesions. Thus in figure 243 although a free effusion was closely simulated, a



FIGURE 243

Encapsulated empyema at left base. See figure 244.

subsequent plate, made after operative removal of the pus, revealed the fixation of the lung to the chest wall along the margin of the empyema. (Fig. 244.) The physical signs in these cases are apt to be ambiguous and may, when taken by themselves, make it difficult to decide between an empyema or a persistent pneumonia with a thickened pleura. The dulness may not be marked, the upper level of the effusion may be horizontal as with a pneumonia and rales may be heard along its borders.



FIGURE 244

Encapsulated empyema at left base after operation, showing adhesion of lung above empyema. See figure 243.

In other cases, and these are the majority, encapsulation of the empyema may at once be inferred from the Roentgen plate. A characteristic example is seen in figure 245 in which the effusion followed the rupture of a lung abscess.

(3) APICAL EFFUSIONS. Empyemas are less often confined to the upper portion of the chest than to the base. An apical effusion is not necessarily a complication of upper lobe disease as it may also follow an inflammatory process in the apex of the lower lobe. Such an origin for the empyema is sometimes indicated by its clinical course and the operative findings, although all the physical signs may have been found over the upper lobe.

The Roentgen and physical diagnosis of an apical empyema in its early stage may be equally difficult. This is especially true of children in whom the signs of fluid and consolidation are so apt to be alike. For this reason apical empyemas may be overlooked for some time until the clinical symptoms render the true condition unmistakable. The Roent-

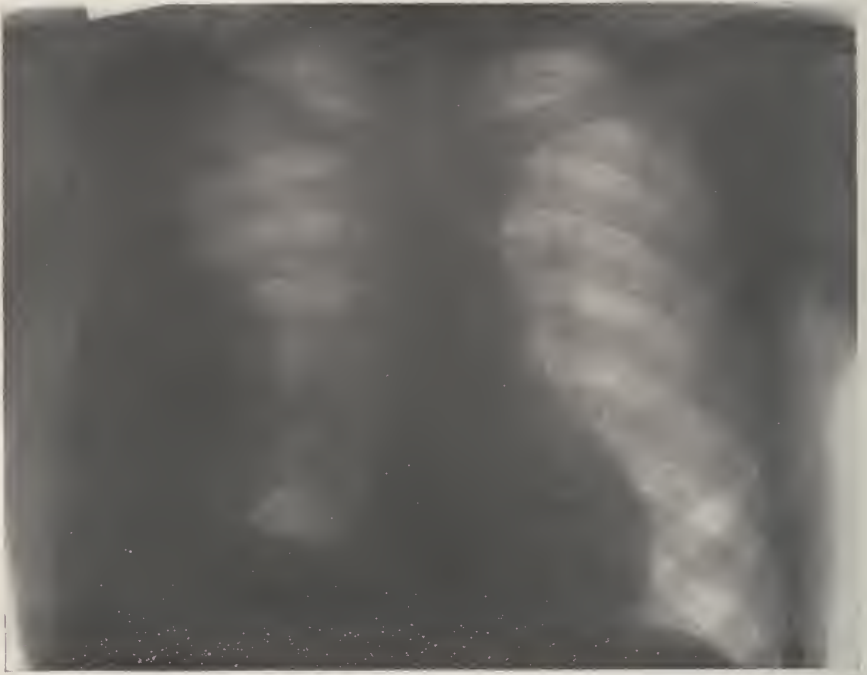


FIGURE 245

Basal encapsulated empyema.

gen shadow may also leave us in doubt, as the appearance of an effusion which is co-extensive with the upper lobe, may be identical with that of a pneumonia. However, sooner or later, a characteristic change occurs in the Roentgen shadow which indicates its true nature. The limited space in the apical region is capable of containing only a small amount of fluid. As the effusion increases it soon extends beyond its limiting adhesions and involves the dependent portion of the chest.

The first evidence of an extension of the apical empyema may be a fibrino-plastic exudate on the neighboring pleura, whose significance as a sign of early empyema we have already discussed. (Fig. 246.) Again, the downward spread of the effusion may be more gradual; the pleuritic shadow slowly extends downward to involve more and more of the chest. The Roentgen picture may exhibit great variety, yet it



FIGURE 246

Encapsulated apical empyema, with fibrino-plastic exudate below, indicating beginning involvement of general pleural cavity.

is scarcely possible to mistake the shadows for those of a consolidation. (Fig. 247.) When such an empyema occupies nearly the entire chest,

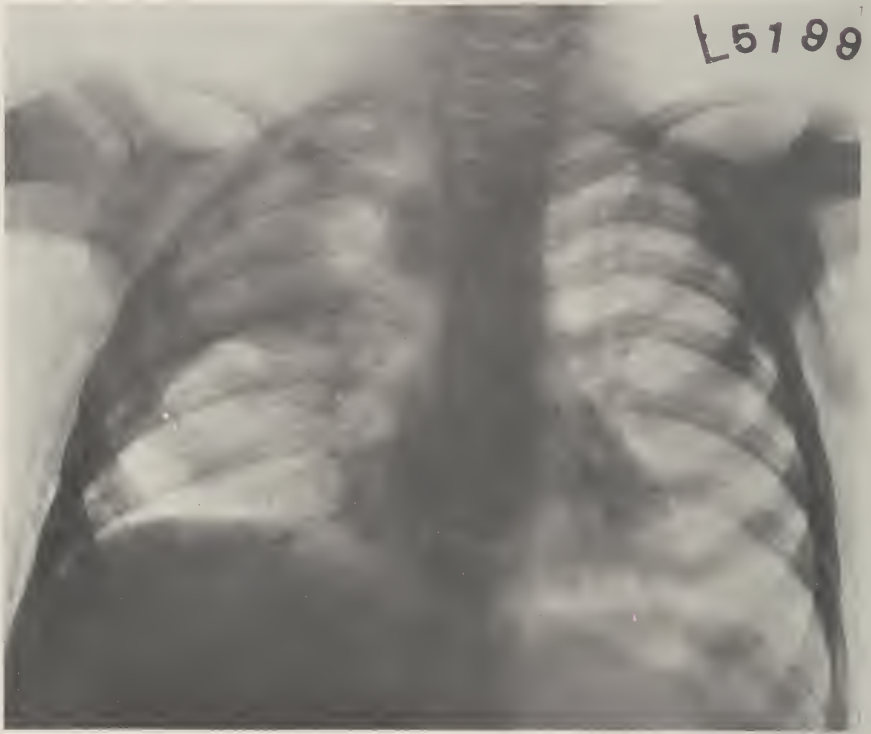


FIGURE 247

Apical empyema, showing extension downward toward base.

so that only the costo-phrenic sinus remains free, the difficulty of distinguishing it from a large free effusion by physical examination is almost insuperable. (Figs. 248 and 249.)

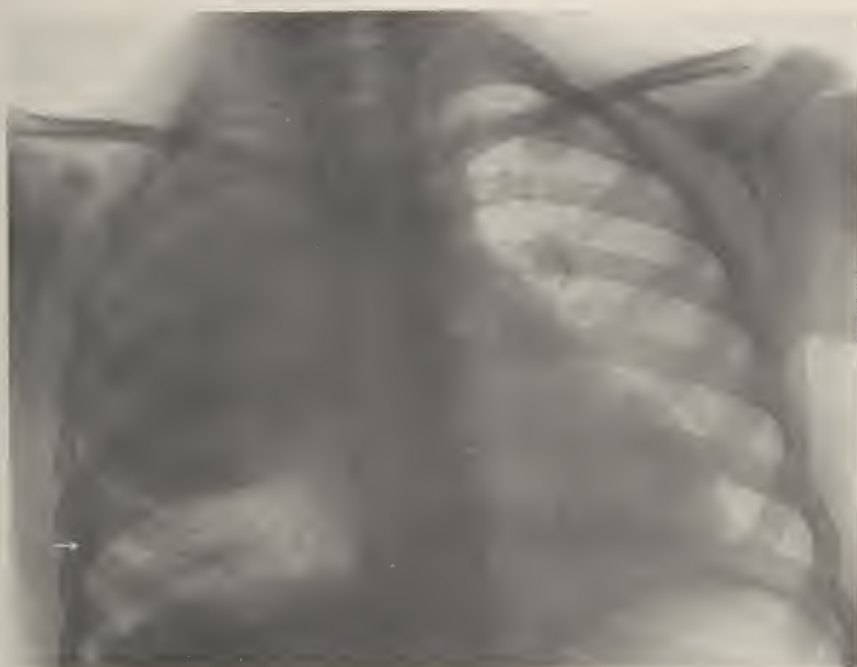


FIGURE 248

Empyema encapsulated in upper two-thirds of chest. Beginning involvement of right base.



FIGURE 249

Encapsulated empyema involving the entire chest with the exception only of the extreme base.

Less commonly we have a sequence of events which is the opposite of that just described, namely, the upward extension to the apex of an encapsulated empyema which began lower down in the chest. Unless the patient has been observed from the beginning and the situation of the physical signs noted, the Roentgen shadows can easily be confused with those of a primary apical empyema. This is illustrated in the following case:

In a young child, one week after the onset of its illness, both physical and Roentgen examination showed an encapsulated effusion, limited to the lower axillary region of the right chest, extending somewhat posteriorly. During the next two weeks the effusion rose to the apex, the Roentgen plate (fig. 250) then showing the apical empyema, and also a downward prolongation of the shadow which represents the original axillary effusion. At operation, there was found a bilocular empyema, one sacculum of which occupied the axillary portion of the chest and the other, the posterior aspect of the lower lobe. Its cause was a ruptured cortical abscess.



FIGURE 250

Encapsulated empyema. In this case, the extension was from below upward, as observed clinically.

The effusion need not surround the lung on all sides but may be situated on its anterior or posterior aspect. The physical examination is better adapted to determine this point than the Roentgen plate. In the case just described all the physical signs of effusion were found posteriorly and, as is so often the case, the percussion note anteriorly over the compressed upper lobe was tympanitic.

(4) MESIAL EFFUSIONS. That portion of the general pleural cavity which borders on the heart is the least common site for encapsulated effusions. Owing to the limited respiratory movement of this part of the chest adhesions often wall it off from the lateral half of the pleural cavity so that it is shielded from infection.

These effusions possess a special interest for the surgeon because they are the occasional cause of puzzling complications after empyema operations. Owing to their unusual situation and the altered physical conditions in the chest consequent on the postoperative pneumothorax, they are only with difficulty found by physical examination. We therefore owe their timely discovery to the Roentgen Ray.

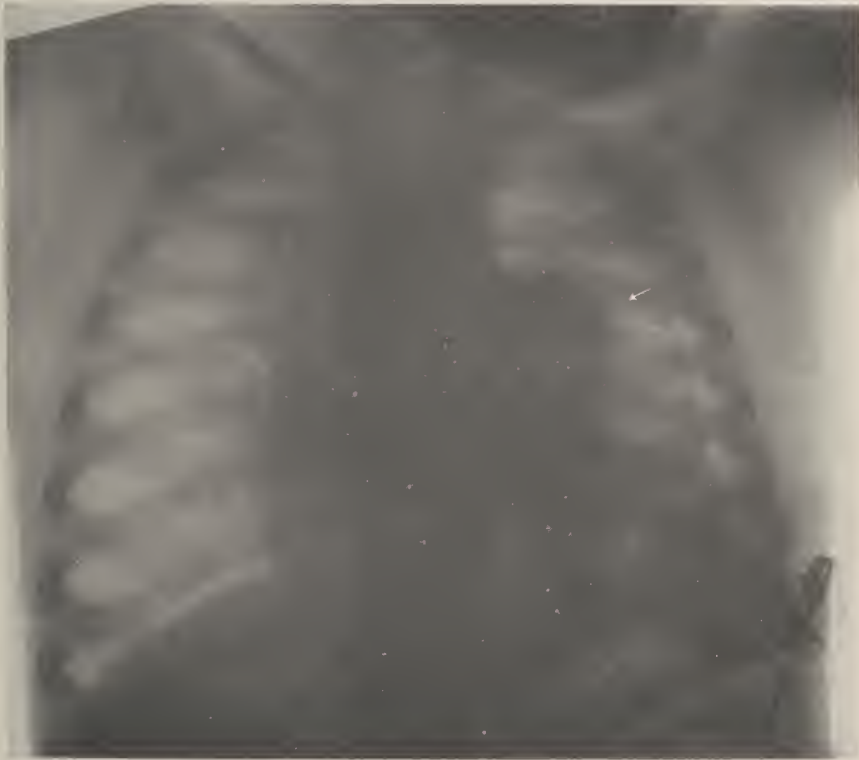


FIGURE 251

Mesial empyema. Shadow in region of pulmonary artery was a residual collection of pus which remained after operation. Note drainage tube in operative pneumothorax. See figure 252.

The problems in diagnosis and treatment involved in these cases are set forth in the following clinical history of a unique case of mesial empyema:

A child two and one-half years old had cough and fever for thirteen days with signs of a large left-sided empyema. A thoracotomy was performed and a large amount of pus, containing staphylococcus aureus was evacuated. Although drainage appeared to be free, high temperature continued for four weeks. At length, during a search for a hidden undrained collection of pus, there was found an area of dulness along the left border of the heart, extending from the 2nd to the 4th ribs. This area of dulness corresponded exactly to the semi-circular shadow which is seen at the left border of the heart in figure 251. An intercostal incision was made at this point and two ounces of pus removed. In the subsequent plate, (fig. 252,) made after operation, this shadow had disappeared. The child promptly recovered.



FIGURE 252

Same case as figure 251 after drainage of mesial empyema.

A mesial empyema, as in this case, may be a residual one and may have existed from the beginning of the illness. On the other hand there are cases in which it is a later development resulting secondarily from an empyema in the lateral half of the chest. We have observed this complication several times after the evacuation of pus from the outer part of the chest and it may be presumed that during the operation the

severance of mesial adhesions permitted the infection to gain access to the hitherto uninvolved mesial part of the pleural cavity. This is illustrated in the following case:

The patient, whose plate is reproduced in figure 253 was operated on and a collection of pus, walled off in the lateral half of the chest, was removed. A few days later he had a chill with high fever. As there appeared to be no retention of pus in the original empyema cavity, a secondary collection was sought elsewhere. On examining the chest there was found an area of dulness along the right border of the sternum in a situation not previously involved. The Roentgen plate proved this to be a collection of fluid surmounted by air in the mesial part of the chest. (Fig. 254.) As is seen, the original empyema had been effectually evacuated leaving a large area of pneumothorax bounded by the adherent lung. There can be no doubt that the surgeon while probing with his finger the limits of the original empyema, broke through the adhesions, thus permitting both infection and air to gain access to the mesial part of the chest.



FIGURE 253

Encapsulated, bilocular empyema. See figure 254.



FIGURE 254

Encapsulated effusion shown in figure 253 after operation. Removal of pus has left a pneumothorax limited mesially by adherent lung. Secondary pyopneumothorax along right border of heart. Arrow points to adherent lung.

(5) **MULTILOCULAR EFFUSIONS.** The adhesions which cause effusions to be encapsulated are also effective, in most cases, in confining them to a limited area of the pleural cavity. For this reason, they may also be described as "localized." Such a localized effusion usually consists of a single collection of fluid which can readily be drained through an opening.

Encapsulated empyemata however, need not be localized. They may occupy a large part of the pleural cavity and may then consist of two or more adjacent sacculations which may or may not communicate with each other. Naturally, the physical signs produced by such sacculations lying next to each other, may be identical with those of a large free effusion. For similar reasons the Roentgen plate will fail to make this distinction. Usually the multilocular character of the effusion is only suspected when a drainage operation is not successful in relieving the symptoms. Under these circumstances, a residual collection of pus may later be found near the apex, which is shut off by



FIGURE 255

Residual empyema at apex after evacuation of basal effusion. Note small air bubble at summit of effusion.

adhesions from the empyema at a lower level which is being drained. For the early discovery of these residual effusions, the Roentgen plate is almost indispensable because their physical diagnosis is made exceptionally difficult by the previous operation. The ease of the Roentgen diagnosis on the contrary is illustrated in figure 255. There is clearly shown a residual emypema at the right apex, which remained after the evacuation of what appeared to be a large free effusion. It gave rise to urgent symptoms after operation, until it was discovered and drained through a separate opening in the chest.

Certain clinical aspects of multilocular effusions may profitably detain us for a moment because of the light which may be thrown on them when they are correlated with the Roentgen findings. The fluid in the various sacculations may be of different characters so that aspiration of the chest may at one point bring forth pus, whereas at another the effusion may be slightly turbid or even serous. Such an unusual circumstance is apt to occur when a lung abscess is near the surface or actually perforates it. There usually results an encapsulated empy-

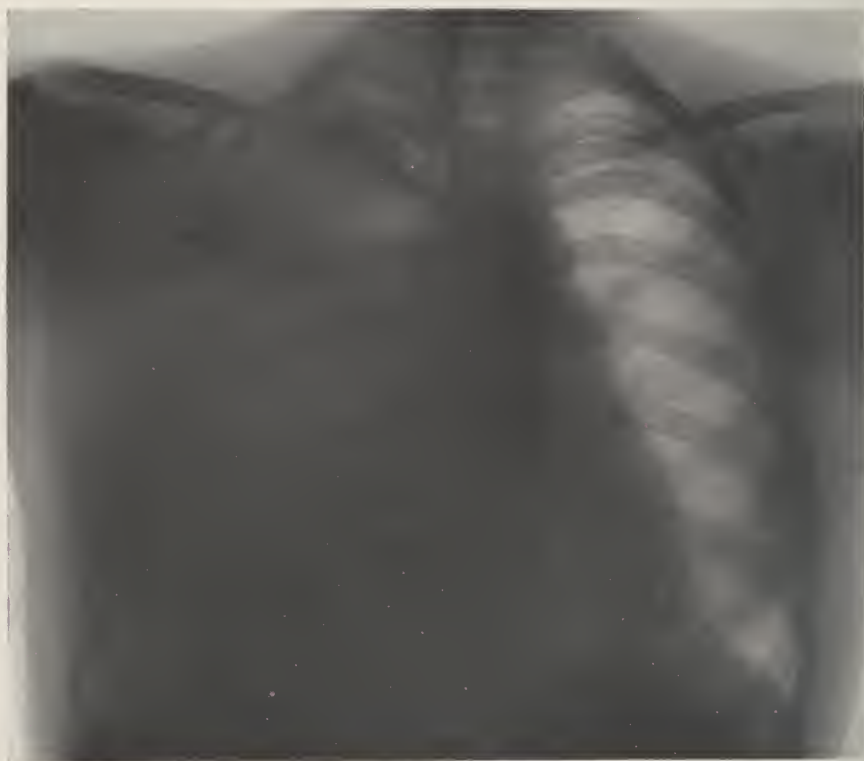


FIGURE 256

Bilocular pleural effusion. The two effusions are adjacent to each other so that they appear to form one large effusion. See figure 257.

ema, localized to the site of the perforation. If the infection spreads beyond the adhesions the neighboring pleura is stimulated to progressive reaction. In the beginning this is fibrino-plastic, it then passes rapidly through the various stages of an inflammatory effusion, from an aseptic serous polynuclear one to a true empyema. When under these circumstances the chest is aspirated over the secondary nonpurulent effusion the original empyema will escape detection until a persistence of the symptoms leads to a search for it. We may illustrate the interesting clinical problem here presented by the following case:

A young woman, some weeks before her present illness, was affected with a succession of furuncles. For one week she had high fever and pain in the right chest. The physical signs were those of a large pleural effusion nearly filling the right chest. The high fever and the blood examination indicated that this was a purulent effusion. However, the fluid aspirated from the base of the chest was clear and sterile. It was significant that it showed a differential count of 90% polynuclear cells. Such an aseptic polynuclear pleurisy is due to one of two causes, either a cortical lung abscess or a near-by encapsulated empyema. There being no evidence of a lung abscess, search was made for a localized collection of pus in the pleura. After repeated aspiration, thick, white pus, containing staphylococcus albus was found in the supraspinous region. Judging from the nature of the infecting organism, we had here evidently an encapsulated empyema at the apex, which was either metastatic or secondary to a furunculosis of the lung, with an aseptic pleurisy of the dependent part of the pleura. At this stage of the disease, owing to a confluence of the shadows of the separate effusions, the Roentgen appearance was that of one large effusion. (Fig. 256.) A few days later the aseptic lower effusion became purulent and a low thoracotomy was performed. After a temporary improvement, and although drainage appeared to be free, the patient once more became acutely ill. It was at once suspected that an apical encapsulated empyema had not been drained by the operation and this suspicion was strikingly confirmed by the Roentgen examination. The plate, (fig. 257) showed that the lower empyema had been emptied. The apical collection, whose outlines had previously been obscured, could now be made out and it was seen that the drainage tube was arrested at its lower limits by the encapsulating adhesions. After a secondary operation this residual collection was evacuated and the patient recovered.

(B) CONCEALED EFFUSIONS. The effusions which we have thus far considered are aptly described as "parietal" in that they are in contact with the chest wall. Although their physical diagnosis is at times difficult, yet because of their superficial situation an approximate diagnosis and localization is usually within the powers of the clinician. This is not the case with so-called "concealed" effusions. They do not come in contact with the chest wall. Here the collection of fluid is buried within one of the recesses of the pleura or it is enclosed between the lung and the adjacent organs. Its physical signs are either absent or so modified by interposed lung that a certain diagnosis is rarely possible. According to their location, we may recognize the following forms of concealed effusions:

- (1) Intrapulmonary,
- (2) Mediastinal,
- (3) Interlobar.



FIGURE 257

Same patient as figure 256 after drainage of lower collection of fluid. There remains a well encapsulated apical empyema just beyond the tip of the drainage tube.

(1) INFRAPULMONARY or diaphragmatic effusions are formed between the lower surface of the lung and the diaphragm. The fluid is retained in this position by adhesion of the edge of the lung to the chest wall or to the diaphragm. It is obvious that the physical signs of such a fluid accumulation will be difficult to interpret. It will especially be necessary to distinguish it from suppurative disease of the liver or the subphrenic space. In figure 258 is shown such a diaphragmatic empyema in a man, whose chief symptoms were cough, remittent fever and copious expectoration lasting some months. At the right base there was an area of dulness extending from the angle of the scapula to the base, where it was flat. The upper line of dulness was convex. Breath sounds were diminished above and absent at the base. At operation there was found a large collection of pus, which rested on the



FIGURE 258

Diaphragmatic or intrapulmonary empyema. See figure 259.

right diaphragm and was enclosed above and at the sides by adherent lung. The relation of the empyema cavity to the surrounding structures may be seen in the post-operative plate. (Fig. 259.)

(2) MEDIASTINAL EFFUSIONS. A collection of fluid between the mediastinum and the lung is undoubtedly the rarest of all effusions. Its development presupposes a fixation of the anterior or posterior margin of the lung, by which the fluid is retained in the mediastinal recess and is prevented from communicating with the general pleural cavity. An effusion in this situation lies in contact with the heart and is entirely enclosed by the overlying lung. For this reason physical signs are not well marked and even when present they are scarcely of a character to suggest a deep seated effusion. We therefore owe the clinical

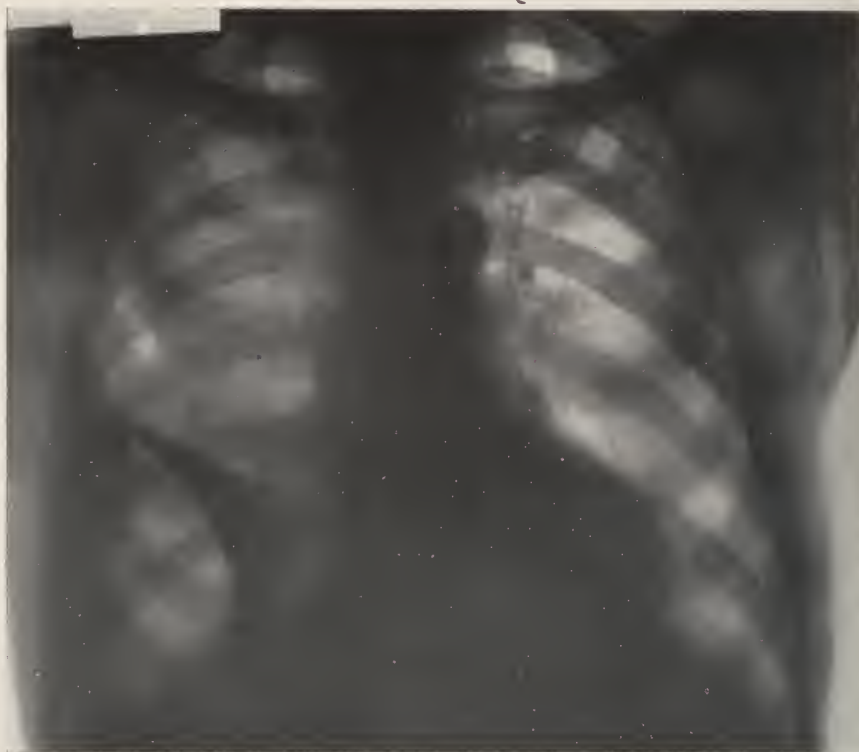


FIGURE 259

Diaphragmatic empyema shown in figure 258, after operation, showing lower margin of lung adherent.

diagnosis of this interesting condition almost entirely to the Roentgen Ray. We may illustrate the Roentgen appearance of mediastinal effusions by the following two cases which also bring out their medical and surgical features.

A woman, twenty-four years of age, had a moderately advanced pulmonary tuberculosis. She was acutely ill for four weeks with fever, dry cough and pain in the left chest. On admission to the hospital she had signs of a small effusion at the left base, which proved to be clear fluid, containing 95% lymphocytes. A week later there was noted an area of slight dullness along the left border of the sternum at the second and third interspaces. There were no breathing changes. The heart was slightly displaced to the right. The Roentgen plate, (fig. 260) was most unusual. In addition to a bilateral upper lobe tuberculosis and an effusion at the left base, there was a dense shadow along the left border of the heart and continuous with the latter. This was presumably due to a mediastinal effusion. The subsequent course confirmed this presumption. After two weeks, all the symptoms subsided and a second plate, (fig. 261.) showed that both the basal and mediastinal effusion has been absorbed. The only evidence which remained of the previous pleurisy was a pleuro-pericardial adhesion at the base which had probably been effectual in walling off the mediastinal effusion. In this case there is every reason to believe that the effusion was of tuberculous origin and it is not improbable that it was secondary to a tuberculous process on the mediastinal aspect of the lung.

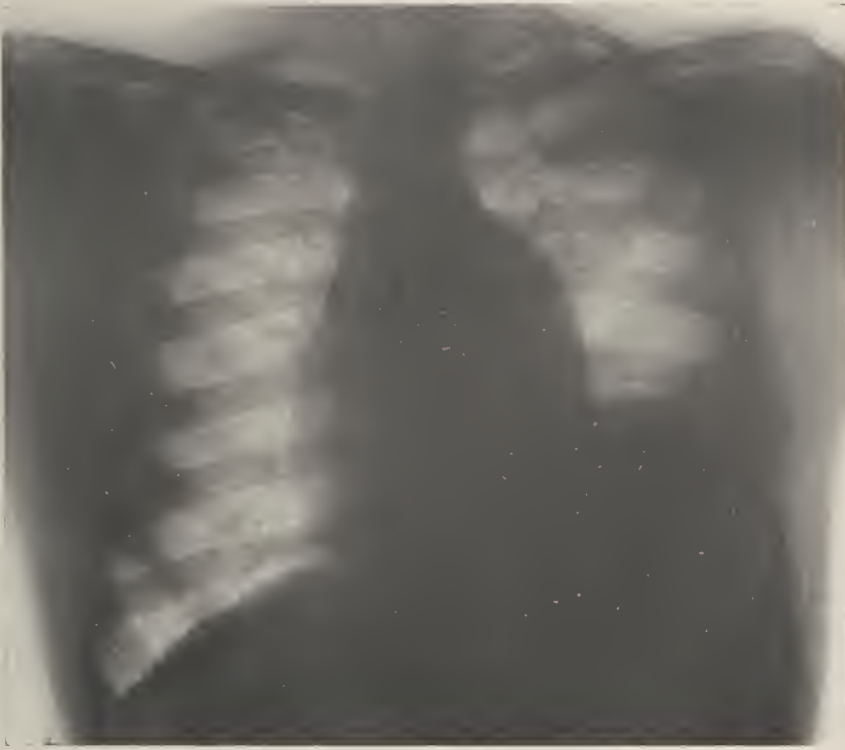


FIGURE 260

Mediastinal effusion with effusion at left base. Upper lobe tuberculosis. See figure 261.

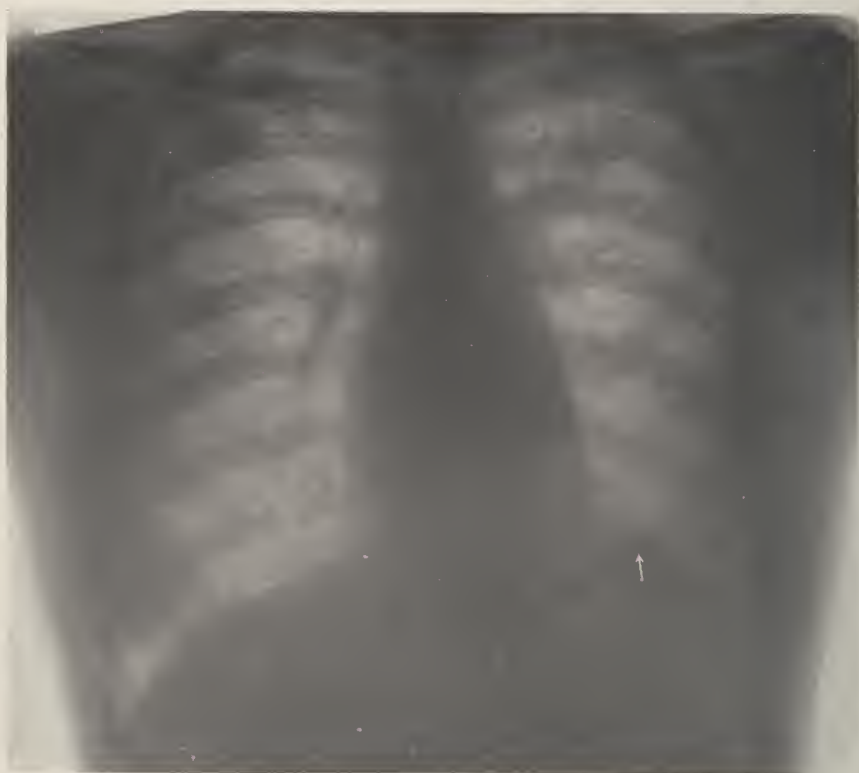


FIGURE 261

Absorption of mediastinal effusion shown in figure 260. Adhesion of left diaphragm to pericardium.

The surgical aspect of mediastinal effusions is brought out in the following case whose complicated clinical features found a counterpart in a most unusual Roentgen picture:

The patient was three months old. For fourteen days previous to admission the child had pneumonia. On admission the temperature was high and remittent and on the left side there were signs of a large pleural effusion which, there was reason to believe, was unencapsulated. At operation a considerable amount of thick pus was evacuated through the usual posterior incision. In spite of apparently free drainage, the temperature continued and the child became increasingly septic. A physical examination at this time disclosed a slight widening of the area of cardiac dulness in its left upper portion, which was presumed to be due to a pericardial effusion. The Roentgen plate, (fig. 262,) taken at this time showed a large cardiac shadow, whose shape seemed to confirm the clinical diagnosis of pericarditis. An operation for the drainage of the pericardial sac was thereupon undertaken. After resecting the left fifth costal cartilage, the anterior margin of the left lung was found to be adherent to the pericardium. The latter was incised but no pus or fluid was found within the pericardial cavity. When, however, the lung was stripped away from the heart there were found, situated between the mediastinal surface of the lung and the heart, six ounces of pus, which had cast the unusual shadow simulating a pericardial effusion. The disappearance of this shadow, after evacuation of the pus is shown in a subsequent plate, (fig. 262). The proximity of these effusions to the heart may make them appear to be a part of it and the distinction between them and a pericardial effusion or a cardiac lesion may be impossible.



FIGURE 262

Mediastinal empyema, before and after operation. Shadow closely resembles that of pericardial effusion.

(3) INTERLOBAR EFFUSIONS. Of all effusions in the chest, excepting only the mediastinal form, the interlobar varieties offer the greatest difficulty in diagnosis and localization. Situated as they are, within the pulmonary fissures and completely enveloped by the lungs, physical signs are either entirely absent or they are so modified by interposed lung tissue, that their diagnosis is at best very uncertain.

The lack of physical signs may also be attributed to the usually small size of these effusions, which is determined by the narrow confines of the fissure.

It is not improbable that many interlobar effusions are clinically unrecognized. This is especially true of the nonpurulent variety which is often latent and finally becomes absorbed. Even interlobar empyemata frequently pass through a period of latency until rupture into the general pleural cavity or into a bronchus draws attention to the pleural cavity.

Interlobar effusions are as rare as they are difficult to diagnose by the usual clinical means. There is little justification for regarding an effusion as interlobar, as is so often done, because the physical signs are atypical and are situated in the region of one of the fissures. It will be found in most cases of this sort that the effusion is in reality encapsulated in the axillary region rather than interlobar.



FIGURE 263

Pneumonia of right lower lobe before development of interlobar effusion. See figure 264.

The clinical recognition of interlobar effusions we owe almost entirely to the Roentgen examination, which reveals accurately their situation within a fissure. An interlobar collection of fluid will lie in the plane of the fissure and will cast a more or less characteristic shadow, depending on its size and situation. It is therefore necessary to bear in mind the topographical relations of the interlobar fissures. The essential points to keep in mind are the following: The upper interlobar fissure, present only on the right side, extends transversely across the chest from the axilla to the sternal end of the fourth rib. Its plane is practically horizontal. The lower fissure begins at the sternal end of the sixth rib, curves upward to the axilla and then ascends sharply upward on the posterior aspect of the lung to the level of the second or third dorsal vertebra.

Interlobar effusions occur most commonly on the right side because of the presence these of the upper interlobar fissure which is their most frequent situation. Only occasionally is fluid found between the upper or middle and lower lobes.

We may clinically recognize three types of interlobar effusions as follows: (1) Metapneumonic; (2) Tuberculous; (3) Idiopathic.



FIGURE 264

Interlobar effusion developing during course of pneumonia shown in figure 263.

(1) METAPNEUMONIC INTERLOBAR PLEURISY: The effusions in the interlobar fissures which occur during the course of a pneumonia, may be either of a serous or purulent character. The former are usually overlooked as they promptly resorb without giving rise to any symptoms. Purulent interlobar effusions complicating pneumonias may occur either alone or in association with an empyema of the general pleural cavity. They cast a characteristic shadow in the region of the fissure which is easy to distinguish from the surrounding pneumonic infiltration. (Figs. 263, 264 and 265.) When such a shadow appears on the Roentgen plate during the course of a pneumonia we need seek no further for the cause of a continuation or a recrudescence of the fever and other symptoms. The following case illustrates the value of the Roentgen examination under these circumstances:

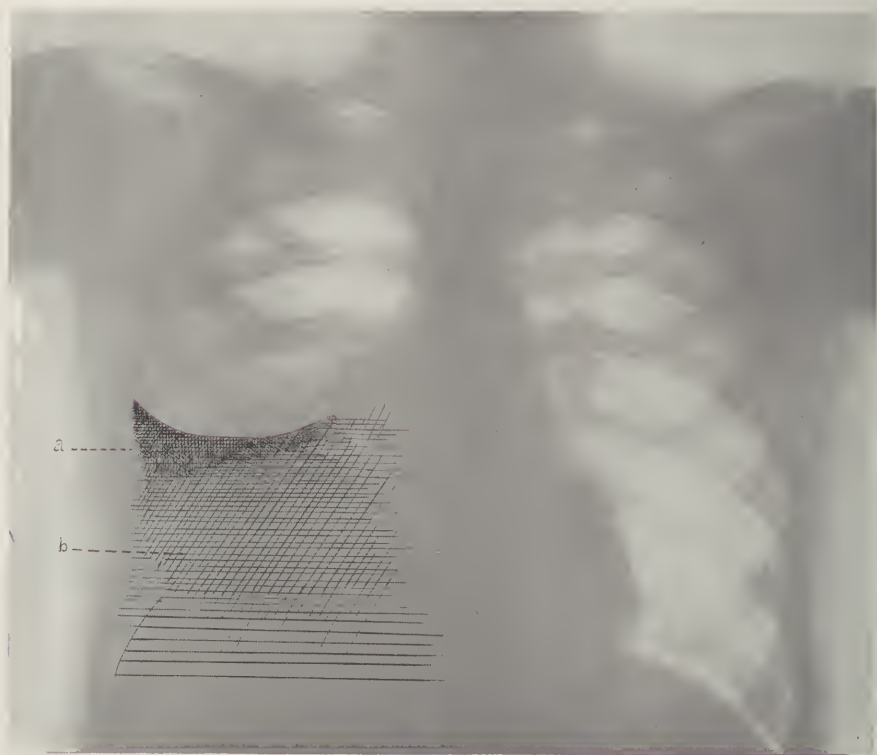


FIGURE 265

Diagram of interlobar effusion complicating lobar pneumonia shown in figure 264.
(a) interlobar effusion; (b) pneumonia.

A child, one year old, continued to have high fever two weeks after the onset of a pneumonia. The physical examination showed only the signs of a resolving pneumonia of the left lower lobe, which did not seem to be an adequate cause for the high temperature. The Roentgen plate, (fig. 266,) however, showed a significant shadow in the region of the right upper fissure, pathognomonic of a pleural exudate. Death ensued in a few days from toxemia. At autopsy a small amount of pus was found between the right upper and middle lobes in the position indicated on the Roentgen plate.



FIGURE 266

Early interlobar empyema on right side; resolving pneumonia at left base.

In figure 267 a similar effusion is shown on the left side, which is very unusual. The lower position of the shadow is here due to the lower situation of the fissure on the left side. Naturally, where so little fluid is present, only a fortunate chance will enable the operator to discover it with the exploring needle, owing to the narrow limits of the fissure. It is very probable that interlobar empyemata, as they increase in size, soon extend beyond it and involve the pleural cavity in a general empyema. It is obvious that the underlying interlobar pleurisy will then be completely obscured by the larger effusion so that it will only be discovered by operation. Of course, at this stage of the disease, it



FIGURE 267

Very rare interlobar effusion on left side. The shadow has a lower position than a right sided effusion, corresponding to the lower position of the fissure.

will not be possible to determine whether the interlobar and general empyema developed simultaneously, as may happen, or in succession. We may illustrate this interesting combination by the following case:

A child of sixteen months contracted measles about one month before admission to the hospital. During the previous two weeks she had cough and slight temperature. When first observed the child was very dyspnoeic. Examination showed flatness in the right chest from the spine of the scapula not quite to the base in the axillary half of the chest. Operation in the eighth space, posterior axillary line, brought to light an encapsulated effusion containing twelve ounces of pus. In addition, on separating the upper and middle lobes, a small amount of pus was found in the fissure. The condition is graphically portrayed on the plate, (figs. 268 and 269,) which was made shortly before the operation. The irregular shadow in the apical and lower axillary regions corresponds to the encapsulated effusion. The small interlobar empyema is distinctly shown as a lenticular shadow extending transversely across the middle of the chest.

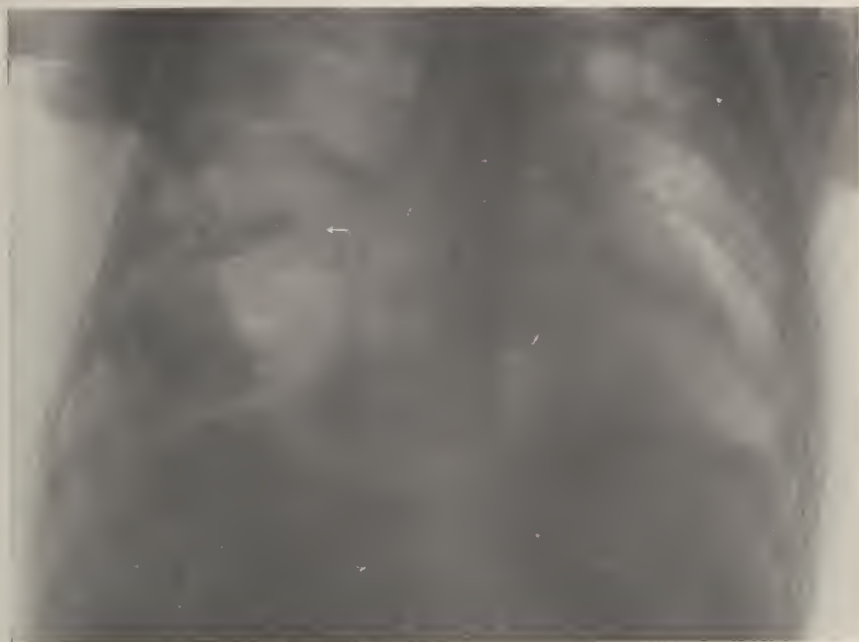


FIGURE 268

Interlobar empyema associated with encapsulated empyema at apex and fluid at base. See figure 269.



FIGURE 269

Diagram of figure 268.

- (a) Encapsulated empyema at apex.
- (b) Interlobar empyema.
- (c) Effusion at base.

A more complicated picture is presented in figures 270 and 271 in which, following pneumonia, an empyema developed not only in the upper fissure but also in the lower one, together with a beginning empyema of the general pleural cavity. One may well marvel at the clearness with which all the details of this complicated empyema are exposed to our view and speculate on the futility of the physical examination to unravel its intricacies.



FIGURE 270

Effusion in upper and lower interlobar fissures following pneumonia. See figure 271.

(2) TUBERCULOUS INTERLOBAR PLEURISY. Effusions of tuberculous origin may develop within an interlobar fissure, as well as in the general pleural cavity. They appear to be more common in children and occur with the greatest rarity in adults. This circumstance is possibly related to the frequent occurrence in children of tuberculous

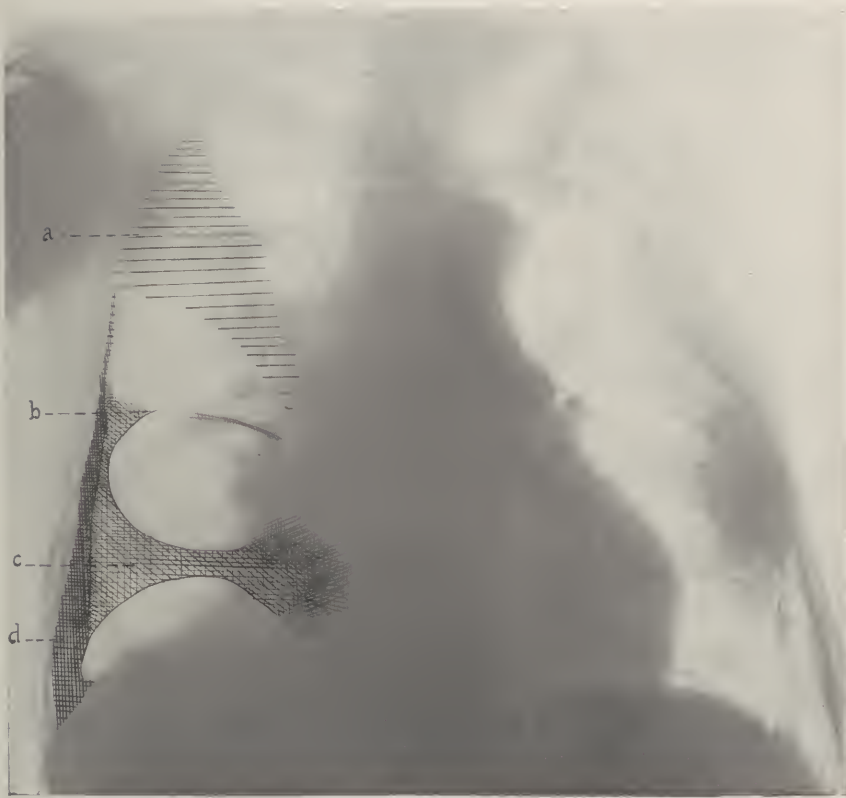


FIGURE 271

Diagram of figure 270.

- (a) Resolving pneumonia.
- (b) Small effusion between upper and middle lobes.
- (c) Effusion between middle and lower lobes.
- (d) Small effusion in general pleural cavity.

infiltrations in the right upper lobe, near the fissure. An interlobar effusion may thus occasionally complicate a hilum tuberculosis in young children (fig. 272) and for some years after its resorption the pleural reflection at the fissure may show a marked thickening.



FIGURE 272

Residual hilum infiltration with a small interlobar effusion.

Naturally, a small interlobar exudate in a tuberculous suspect is easily overlooked and it may not be until the general pleural cavity is involved, that attention is drawn to it. The long period during which such a pleurisy may remain latent, is well brought out in the following case:

A child of eight and one-half years, for three months had headache, fever, weakness and cough. Six weeks before examination she developed pain in the right side with an increase of the cough. The child was pale, undernourished and was apparently toxæmic. There were signs of fluid in the lower third of the right chest. Blood count, 16000 leucocytes; 63% polynuclears. Only a small amount of lymphocytic fluid could be aspirated. On the plate, (fig. 273) may be seen an interlobar effusion which appears to communicate with a small exudate in the general pleural cavity. The clinical facts all point to the tuberculous nature of the pleurisy. It is very probable that the onset of the pleural symptoms was coincident with an extension of the inflammatory process to the general pleural cavity.



FIGURE 273

Interlobar effusion, probably tuberculous. Small effusion in general pleural cavity communicating with interlobar effusion.

In the following case also, the tuberculous nature of the effusion, which was very unusual in situation, cannot be open to doubt:

The child of twenty-one months had pneumonia four months before. Since that time cough was persistent with slight rises of temperature, usually in the evening. Latterly, a number of tuberculous nodes appeared in the neck. The patient appeared only slightly ill. The physical signs were puzzling and pointed to a pleural effusion in an unusual situation. Posteriorly, there was dulness from the apex to the angle of the scapula; below this and extending to the axilla, the note was tympanitic. Anteriorly also there was a tympanitic note. The breath sounds behind were bronchial in the upper chest and broncho-vesicular below. The Pirquet test was strongly positive. A needle was inserted posteriorly below the angle of the scapula, but no fluid was obtained. A deep puncture in the axilla however, brought out a considerable amount of gelatinous fluid, which contained no bacteria. The physical examination suggested a deep seated effusion covered by compressed lung, giving rise to a tympanitic note. The Roentgen plate, (fig. 274,) shows a well encapsulated collection of fluid which is limited below and laterally by the lower fissure. The plate may be regarded as fairly typical of a large effusion between the upper and lower lobes.



FIGURE 274

Large effusion between the upper and lower lobes. Probably tuberculous.

(3) IDIOPATHIC INTERLOBAR PLEURISY. Occasionally an interlobar effusion is found unassociated with existing or previous pulmonary disease. The etiology of these cases is obscure and they may perhaps be regarded as primary interlobar effusions, due to the virus of rheumatism or other infectious agents which are responsible for pleurisies occurring in the more usual situations. In some cases they are entirely latent clinically; others run their course as acute respiratory infections which, because of an absence of physical signs, are mistakenly regarded as deep-seated pneumonias.

For example, following a mild respiratory infection a patient developed symptoms of exophthalmic goitre. On the Roentgen plate of the chest made to determine the size of the heart, an unsuspected effusion was found in the right upper fissure. (Fig. 275.) The patient had no pulmonary symptoms and in two weeks the abnormal shadow had disappeared. A more acute form of this disease is illustrated by the following case:



FIGURE 275

Interlobar effusion.

A child of three years had cough, dyspnoea and high fever for ten days. Physical examination of the lungs was entirely negative. Yet on the plate, (fig. 276,) there was a fairly large collection of fluid between the upper and middle lobes. After a few days the temperature came to normal, the child rapidly improved and the shadow disappeared. We were here evidently dealing with an interlobar pleurisy, whose acute inflammatory character was further attested by the high leucocyte count, which was 22000, polynuclears 80%. At no time were dulness or rales to be heard over the affected area of the chest. This fact and the peripheral position of the shadow will serve to exclude central pneumonia as a cause of the condition.

ENCAPSULATED EFFUSIONS WITH PNEUMOTHORAX. The presence of air in addition to fluid within a pleural sacculation adds measurably to the difficulties of the physical examiner. Whereas the larger collections of air and fluid in the chest betray their presence by such characteristic signs as tympany and succussion, the smaller amounts of air, which are usually found with sacculated effusions, are easily overlooked. Moreover, an area of pneumothorax situated above



FIGURE 276

Acute pleurisy with effusion in right upper interlobar fissure.

a small effusion will render accurate localization of the latter difficult, by masking its dulness. While the physical examiner thus finds his difficulties multiplied, the presence of air in the chest is a fortunate circumstance for the Roentgenologist and it is to him that we owe our intimate knowledge of his condition. Whenever air and fluid coexist in the chest, the latter acquires a horizontal level, the shadow of which is one of the most distinctive features of the Roentgen plate.

We commonly associate the condition of pyopneumothorax with pulmonary tuberculosis, yet it is surprising to note the frequency with which encapsulated collections of air and pus are encountered with pyogenic disease of the pleura. Aside from tuberculosis, perhaps their commonest cause is the extension of a lung abscess to the pleural surface or its actual perforation. This perforation in the majority of cases is for some time preceded by a fibrinoplastic pleurisy so that the subsequently developing empyema is encapsulated. Its communication with a bronchus permits the free access of air to the effusion. This complication of a lung abscess or a bronchiectatic cavity may develop during the early acute stage of the disease or it may be a late and terminal event. In figure 277 is shown a localized pyopneumothorax



FIGURE 277

Encapsulated pyopneumothorax secondary to ruptured lung abscess. The fluid has a horizontal level and is walled off by dense pleural adhesions. (arrow).

which occurred during the course of a gangrenous abscess of the lung, eighteen days after the onset of a pneumonia. The encapsulation of the fluid below is clearly shown. Above and laterally pleural adhesions wall it off from the general pleural cavity. At operation, a small perforation was found on the posterior surface of the lung.

The frequent pleural adhesions which complicate pulmonary tuberculosis will occasionally confine to a limited region of the chest the effusions which follow a spontaneous pneumothorax. Usually the fluid is walled off in the axillary part of the chest. At times it may have an unusual situation, as in figure 278 where it is supported in the middle of the chest by the adherent lower lobe. Among the rarer causes for an encapsulated seropneumothorax is the perforation of a tumor nodule in the lung. The small collection of air and fluid in the right upper lobe in figure 230 was found in a case of miliary carcinosis of the lung.

In contradistinction to the cases we have thus far discussed, in which the air is derived from the lung through a perforation, we must consider those in which the air is introduced from without, either dur-

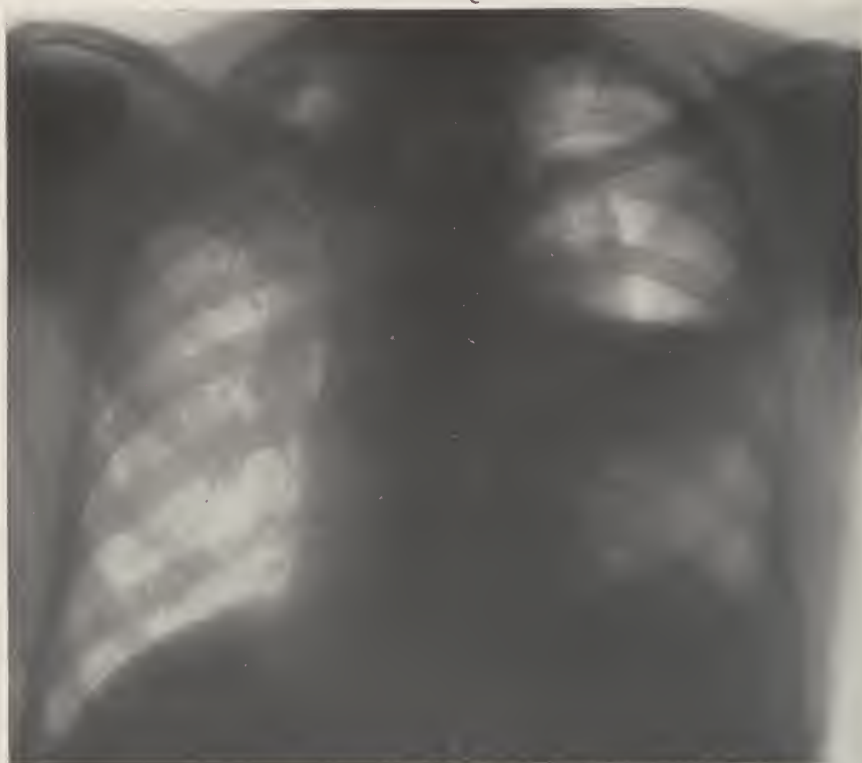


FIGURE 278

Encapsulated pyopneumothorax; pulmonary tuberculosis. Note collapsed upper lobe containing cavity. The fluid occupies the middle third of the chest and shows a horizontal surface due to the air above it.

ing operation or exploratory aspiration of the chest. By this means the shadows of sacculated effusions, which might otherwise be difficult of interpretation acquire a distinctive appearance. Thus, the mesial empyema in figure 254 followed the evacuation of the axillary effusion shown in figure 253. The communication of this effusion with the outer wound was responsible for its horizontal upper level which removed all doubt as to the nature of the shadow. It is not unusual for a small amount of air to be drawn into the chest during exploratory aspiration. This is more apt to occur in encapsulated effusions because the lung, being bound down by adhesions is prevented from expanding after the removal of the fluid. The appearance of such a partial pneumothorax surmounting a fluid level after aspiration of the chest is therefore at times an evidence of sacculatation of the fluid.

The phenomenon of fluid levels in the pleura is of the greatest value in the diagnosis of multilocular effusions. When a number of sacculations lie adjacent to each other the dulness of the individual saccula-

tions becomes merged in such a way that a large single effusion is simulated. If in such a case the chest is aspirated the operator will not be able to evacuate the effusion by a single puncture. It will be necessary to insert the needle separately into each sacculation. When a Roentgen examination is made after such multiple punctures, the separate loculi may be found partly filled with fluid and air, so that their size and situation can be defined. The following case is an instructive example:

Following pneumonia the patient, a young woman, presented the physical and Roentgen signs of a large effusion in the left chest. The chest was aspirated at three points posteriorly and each time from 50 to 100 cc. of clear fluid were removed. A plate made the next day, after air had apparently entered the plural cavity, showed three fluid levels of various lengths, situated one above the other. The vertical dimensions of the sacculations could be accurately determined from the Roentgen plate made in the lateral recumbent position, consequent on the shifting of the fluid. (Fig. 279.) The sequel of this case is of some interest. The fluid, presumably as a result of frequent aspirations became infected with anaerobic organisms. The physical signs then became those of a large unencapsulated pyopneumothorax. Evidently under the influence of the putrid infection, the adhesions were dissolved and the various sacculations coalesced into one large effusion, as is shown in figure 289.



FIGURE 279

Encapsulated pyopneumothorax. Patient in lateral recumbent position showing three fluid levels. See figure 289.

SECONDARY CHANGES IN THE CHEST ASSOCIATED WITH PLEURAL DISEASE

Some of the classical physical signs which result from disease of the pleura are due to mechanical effects on the various intrathoracic structures. The position of the heart and mediastinum, the volume of the chest and the movement of the diaphragm are all under the scrutiny of the Roentgen Ray, which is eminently fitted to register these secondary changes and to supplement by an accurate objective method the findings of the physical examination. Roentgen studies are in the main in agreement with commonly accepted ideas in regard to the effect of effusions on adjacent organs. Pleural effusions of moderate or large size frequently cause a displacement of the heart to the opposite side. This is especially true of noninflammatory effusions, such as the transudates of cardiac or renal disease. It is equally true of the rapidly forming large empyemata in young children, in whom the heart may suffer an extreme displacement. (Fig. 280.)

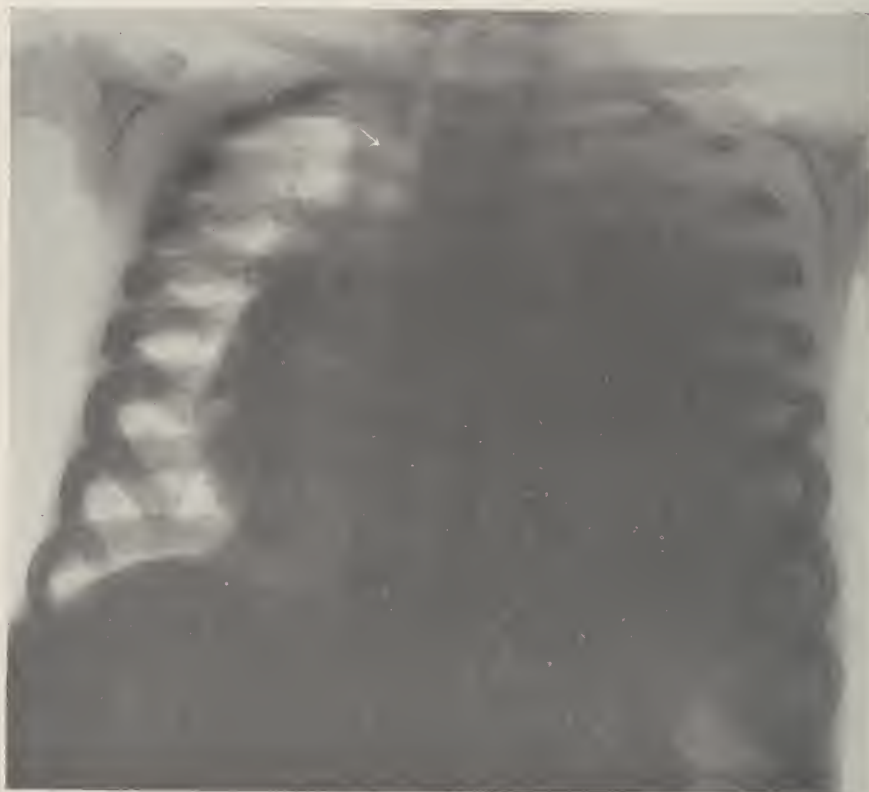


FIGURE 280

Large empyema in child. Note separation of ribs and displacement of heart and trachea to right. (arrow.)

It is however, surprising to note the frequency with which the heart is not at all affected in its position by the presence of fluid in the pleural cavity. Even a very large collection of fluid may cause no malposition of the heart. If we inquire more closely into this phenomenon, it will be found that cardiac displacement depends not only on the size of the effusion but also on the nature of the pathological process which causes it. Thus with empyema, especially in adults, even if it be large, the heart is frequently found in its normal position. Evidently other causes than the pressure of the fluid on the heart must be operative. In the first place, with inflammatory exudates the mediastinum may be fixed by adhesions so that it and the heart are not easily displaced. Secondly, lessened respiratory movement of the lung and consequent atelectasis, due to the inflammatory process, will tend to displace the heart to the same side and thus counteract the direct pressure effect of the effusion. These opposing forces may be so well balanced that in a large percentage of empyemata in adults, the heart appears to maintain its normal position. The paradoxical phenomenon of a displacement of the heart to the pleuritic side may occasionally be seen, even with an effusion of fair size. Here again we must assume a reflex immobility of the lung with atelectasis, whose effects are sufficient to overcome the pressure of the fluid and the elastic pull of the opposite lung. A similar phenomenon may at times be seen in pleurisies without effusion or in the early stages of empyema, before effusion has occurred. The reflex immobility of the chest and the decrease in the volume of the lung may cause a noticeable displacement of the heart toward the affected side.

When we observe on the Roentgen plate these unusual displacements of the heart, which are so much at variance with traditional teachings, we are impressed with the minor role played in their causation by the effusion itself. One is inclined rather to seek an explanation for them in variations of the elastic tension of the lungs and of the intrathoracic pressure.

The influence of encapsulated effusions on the position of the heart presents some points of interest. Whether the heart is displaced or not seems to depend entirely on the position of the encapsulated fluid with respect to the heart borders. When sacculated effusions are situated in the lateral portion of the chest or at the apex, the heart maintains its usual position. If, however, the fluid is in the mesial portion of the chest and is applied to the cardiac surface so that it can exert direct pressure on it, the heart will be displaced. Thus the large axillary effusion in figure 253 had no influence on the position of the heart, whereas the small effusion seen in figure 243 which was close to the apex of the heart caused a distinct dislocation of the latter. After

the evacuation of the fluid the heart resumed its normal position. (Fig. 244.) In a similar way the mediastinal effusion in figure 260 by virtue of its position close to the heart, caused its displacement.

Changes in the conformation of the thorax secondary to effusions can be estimated with great accuracy on the Roentgen plate. In harmony with clinical experience we find in rapidly forming effusions, especially in young children, a separation of the ribs with a widening of the intercostal spaces and an increase in the volume of the affected side. (Fig. 279.) However, the Roentgen findings reveal in many cases a surprising divergence from the accepted clinical view in respect to these thoracic changes. A separation of the ribs is frequently absent in large purulent effusions and in empyemata of moderate size it is practically never found. On the contrary, in a considerable percentage of cases, the ribs are actually drawn closer together so that the affected half of the chest is smaller than the healthy side. Nor need we look upon such a retraction of the chest, as is frequently done, as evidence of an encapsulation of the empyema. Although it is true that retraction of the chest wall is often associated with encapsulated effusions, it is also found with free effusions of a purulent character. It may be regarded as a result of the reflex rigidity of the intercostal muscles, similar in its mechanism to the abdominal rigidity of peritonitis.

CHAPTER XVI

Pneumothorax

The presence of air in the pleural cavity even in small quantities may readily be detected by Roentgen examination. Depending on the amount of air and the presence or absence of pleural adhesions, the lung recedes from the chest wall for a variable distance; between the margin of the retracted lung and the chest wall there is a homogeneous air space, situated usually in the apical and axillary regions, which on the Roentgen plate is characterized by an absence of the pulmonary markings. (Fig. 281.)

The great variety in the size and shape of pneumothoraces is determined by two factors, namely, pleural adhesions and the condition of the lung. When there are no adhesions and the lung is normal, the



FIGURE 281

Spontaneous pneumothorax. Right lung concentrically retracted, showing separation of upper and lower lobes at arrow.

latter is contracted concentrically as the amount of air increases, so that finally it forms a small dense globular mass at the root of the lung. (Fig. 292.) Rarely the air gains access to the mediastinal portion of the chest, (fig. 282) and at times it may insinuate itself within the interlobar fissures so that the individual lobes become visible. (Figs. 281 and 292.)

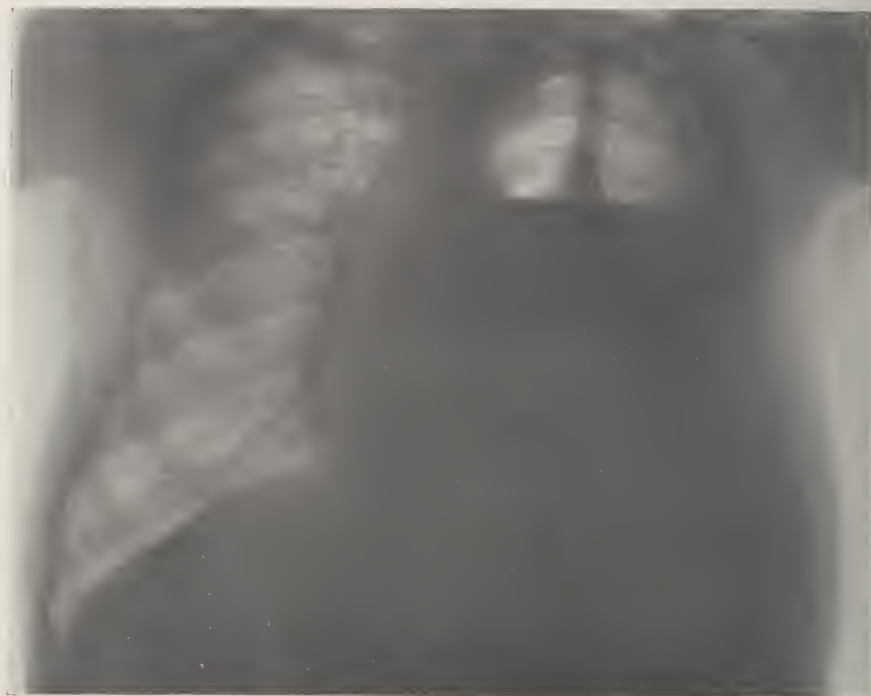


FIGURE 282

Pyopneumothorax; lung adherent in midclavicular line; air has gained access to mediastinal pleura.

The compressed lung becomes progressively more dense until it finally has the opacity of a consolidation. Such a complete collapse however, is not the rule, as either infiltration of the lung or firm pleural adhesions will retain it in contact with the chest wall at one or more points. The commonest situation for these adhesions is at the apex, which is evidently due to the frequency of apical fibroid changes resulting from chronic tuberculosis. For this reason, the lower part of the lung is often found compressed, whereas the upper lobe remains adherent. In some cases the adhesions are extensive (fig. 283); in others

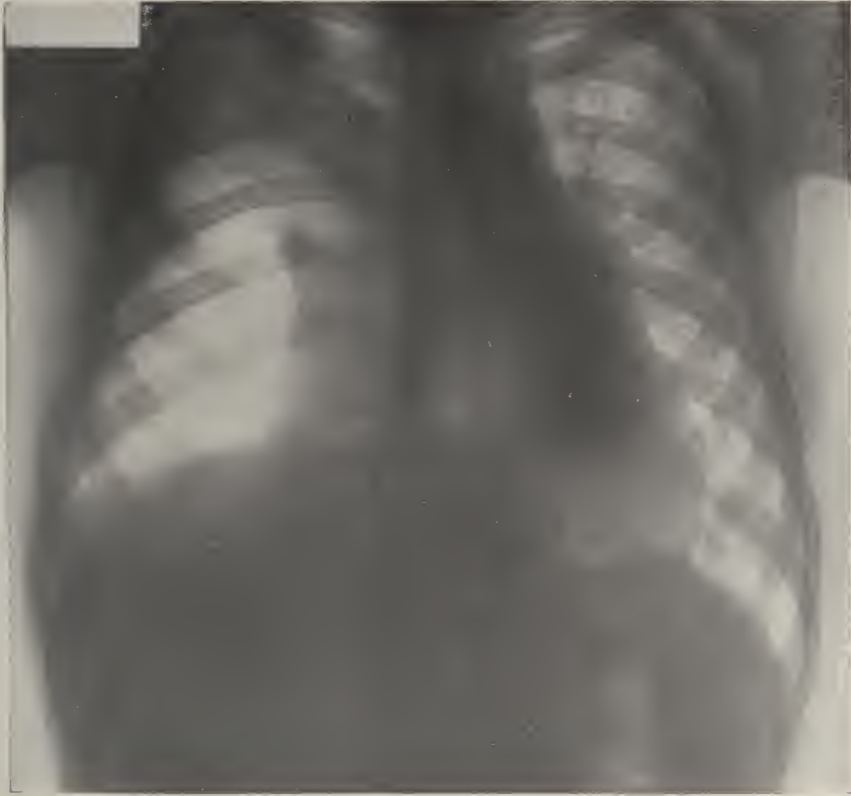


FIGURE 283

Pneumothorax. Upper lobe firmly adherent. Hernia of mediastinum to left side due to extreme pressure of pneumothorax. Heart displaced to left.

(figs. 284, 285 and 286) they may be localized to a small area. Less commonly the apical region may be quite free and the lung may be adherent at its middle (fig. 287) or even at the base.



FIGURE 284

Almost complete pneumothorax, right side. Lung adherent in infraclavicular region. Compensatory engorgement of left lung.



FIGURE 285

Complete pneumothorax, showing compression of lung. Diaphragm depressed and ribs separated. Lung adherent at apex.



FIGURE 286

Spontaneous pneumothorax with miliary tuberculosis of lungs. Apical adhesion.



FIGURE 287

Artificial pneumothorax. Lung completely collapsed except at middle where a few adhesions are seen. Heart displaced to right. Note engorgement of right lung. Depression of diaphragm.

Displacement of the heart is a more regular phenomenon with pneumothorax than with pleural effusions. The loss of tension of the collapsed lung upsets the balance between the two halves of the chest so that the heart is drawn away from the affected side. This is true even though the intrapleural tension be negative. Whenever the pressure increases until it becomes positive, as in cases of tension pneumothorax, the displacement of the mediastinum may be extreme. In fact, under these conditions, there may result a hernia of the mediastinum to the normal side. (Fig. 283.) It is in these cases that the intercostal spaces are much widened by the increased volume of the thorax and the diaphragm is depressed. (Figs. 287 and 288.) There



FIGURE 288

Extensive bilateral tuberculosis with spontaneous pneumothorax under tension.
Separation of ribs and marked depression of diaphragm.

may also be observed on the fluoroscopic screen an inspiratory displacement of the mediastinum to the side of the pneumothorax and a paradoxical upward excursion of the diaphragm.

A pneumothorax produces profound changes both in the lung on the affected side and in the opposite one. The compressed lung acquires a denser shadow which one must not confuse with the infiltrations of disease. Small lesions in the collapsed lung may thus readily be obscured. Occasionally collapse of the lung operates to some advantage in diagnosis. Thus, in a tuberculous or suppurating lung, a cavity frequently becomes more distinct when the pulmonary tissue about it is slightly compressed by the pneumothorax. (Fig. 81.) Marked changes may also be observed in the unaffected lung. When there is an extensive pneumothorax, this lung must perform the function of both and accordingly it is over-supplied with blood. This results in a marked accentuation of the vascular markings, (fig. 287) which must not be confused with disease of the lung.



FIGURE 289

Later stage of case shown in figure 279. Pyopneumothorax.

A number of interesting complications are occasionally associated with pneumothorax which are visible on the Roentgen plate. The most common of these is a pleural effusion, whose characteristic horizontal level is an unmistakable sign of air in the pleural cavity. (Fig. 289.) Usually the fluid is unencapsulated, as in tuberculous cases; yet there are numerous instances, especially in cases of suppurative lung disease in which the fluid is walled off by adhesions so that one (fig. 277) or more collections of fluid and air are found. When much fluid with air is present in the pleural cavity, it may depress the diaphragm so that it presents a downward convexity. Under these circumstances there may be visible on the fluoroscopic screen a paradoxical upward movement of the fluid level during inspiration. Another frequent complication which occurs especially after induced pneumothorax, is subcutaneous emphysema, in which the air may be seen to infiltrate the tissues for a



FIGURE 290

Pyopneumothorax complicated by pneumopericardium. Arrows indicate pericardial membrane raised from the heart by air in pericardium. Patient in lateral recumbent position. Air and fluid in left chest, below.

considerable distance. A very rare complication of pneumothorax is a perforation of the pericardium as a result of which the unique picture of pneumopericardium is produced. (Fig. 290.)

Roentgen studies have done much to extend our knowledge of pneumothorax. It is no exaggeration to state that before the introduction of the Roentgen Ray the majority of cases, especially of partial pneumothorax, were unrecognized. It has been shown that little dependence can be placed on the classical syndrome of sudden pain, collapse and cyanosis, as these symptoms are absent in many cases. Physical signs also, except in the presence of much air, are very ambiguous. For the diagnosis of the small pneumothoraces, which are so frequent in pulmonary tuberculosis and the local pyopneumothoraces of lung abscess, dependence must be placed entirely on the Roentgen Ray as physical examination rarely discovers them.



FIGURE 291

Irregular pneumothorax, due to pleural adhesions, following pneumonia, resembling cavities in lung. Complete restoration to normal in a few weeks.

The distinction between a localized pneumothorax and a large pulmonary cavity which is often uncertain clinically, presents little difficulty to the Roentgenologist. Cavities which could be thus confused are usually the large tuberculous excavations in an upper lobe. In these cases, although very little pulmonary tissue may remain about the cavity and although the latter may superficially resemble a pneumothorax, the thickened wall of the cavity can practically always be seen. Irregular encapsulated pneumothoraces however, as in figure 291, present much difficulty in diagnosis unless the clinical history is available.

ARTIFICIAL PNEUMOTHORAX

Since the introduction of the operation for artificial pneumothorax, the Roentgen examination of the chest has acquired a practical importance as an adjunct to therapy. Although an artificial pneumothorax may be induced and maintained without the control of the Roentgen Ray, it is so performed at the cost of scientific exactitude and also at some risk. In the first place, the preliminary examination is of the greatest value in determining the extent of the disease, whether both lungs are involved and which side is the more advanced. It will therefore determine the side on which the pneumothorax should be induced. In the second place, the examination may locate adhesions or thickening of the pleura and may thus guide the operator in choosing the site of puncture. Further, after the gas had been introduced the Roentgen examination is almost indispensable to ascertain with any degree of exactness when the lung is completely collapsed. Only by means of it will the operator become aware of firm, inseparable adhesions which will discourage any attempts further to compress the lung. The Roentgen examination will also control the time of the re-injection of the gas and finally it will very early disclose the development of an effusion which may make it inadvisable to attempt a further injection.

In many cases these purposes are better served by the fluoroscopic than by the plate examination. On the fluoroscopic screen one obtains a better impression of the firmness of adhesions and the likelihood of severing them. Frequently a single dorso-ventral plate gives an entirely erroneous impression of the amount of air introduced and the degree of the collapse of the lung. Thus it is not an uncommon experience to find on the plate evidence of only a small pneumothorax in the axilla. The fluoroscopic examination in such cases may show a considerable amount of air in the posterior portion of the chest, whose presence is obscured on the plate by the overlying lung.

Only a few illustrations of the Roentgen appearance of artificial pneumothorax in its various phases need be presented, as they differ little if at all, from spontaneous pneumothorax. In figure 292 are seen



FIGURE 292

Two stages in artificial pneumothorax, with complete collapse of lung. Note separation of lobes and later depression of diaphragm and cardiac displacement with increasing pressure.

two stages in the progress of a therapeutic pneumothorax terminating in a complete collapse of the lung. In figure 287 the gas is under such great tension that the mediastinum and heart are displaced for a considerable distance into the opposite chest. Occasionally the Roentgen plate discloses the reason for the failure of the injected gas to effect a collapse of the lung. Thus in figure 397 because of a low insertion of the needle, which penetrated the diaphragm, considerable gas was injected into the peritoneal cavity, where it is visible. In other cases, an extensive subcutaneous emphysema offers a ready explanation for an unsuccessful injection.

CHAPTER XVII

Tuberculosis of the Pleura

As is well known, inflammation of the pleura is a frequent manifestation of the tuberculous infection. The commonest form of pleural disease is associated with a tuberculous involvement of the lung and consists of a fibrino-plastic exudate which later organizes and gives rise to the pleuro-phrenic adhesions which are such a familiar feature of the Roentgen plate in these cases. A thickening of the pleura in other situations than at the base is not readily demonstrable on the Roentgen plate unless it is considerable, as the rays traverse it readily. At the interlobar fissure however, on the right side, which is a favorite site of pleural thickening, the pleura at the anterior edge of the upper and middle lobes becomes visible as a sharp linear shadow which traverses the chest from hilum to axilla. It will be found on routine examination of the lungs of adults that from five to ten percent of all individuals show this shadow more or less distinctly. In all probability it is dependent on a previous tuberculous infection in this portion of the lung or conceivably it may stand in relation to the neighboring tuberculous lymph nodes at the hilum.

The extreme thickening of the pleura associated with chronic fibro-caseous tuberculosis is of course very common and it contributes considerably to the density of the shadows found in advanced cases of this disease.

Next in frequency to pleural adhesions and thickenings are serous effusions. Although clinically the tuberculous origin of pleural effusions is taken for granted in a large percentage of the cases, it is only occasionally, as in figure 260 that it is possible to demonstrate a coincident lesion in the lung. As a rule, the effusions do not differ in appearance from other forms of pleurisy, be the exudate serous or purulent.

Among the rarer forms of pleural tuberculosis are localized deposits which, like the lesions in the lungs, may undergo caseation and calcification. The Roentgen shadows are characterized by great density and owing to their proximity to the surface of the chest, by a sharpness of definition not seen in more deeply seated processes. These features of pleural tuberculosis are brought out in the calcified deposits in the right lower chest shown in figure 293. In figure 294 the pleura is studded with numerous large tubercles which are associated with a general thickening of the pleura and a retraction of the chest wall.



FIGURE 293

Calcified tuberculosis of the pleura. Calcified bronchial lymph nodes.



FIGURE 294

Chronic tuberculosis of pleura with much thickening and calcified tubercles.

Bizarre forms of pleural tuberculosis with calcification as in figure 295 suggest the "Perlsucht" of cattle and the likelihood of a bovine infection.



FIGURE 295

Bizarre form of calcified tuberculosis at left base. Chronic tuberculosis at left apex.

Finally, miliary tubercles in rare cases become visible on the pleura during the course of an acute miliary tuberculosis. Such a group of tubercles is seen at the extreme left base in figure 296. They were discovered in a patient with tuberculous meningitis in whom, shortly before death, showers of fine rales were heard at the base of the left lung. It will be noted that the lungs themselves show no evidence of miliary tuberculosis.

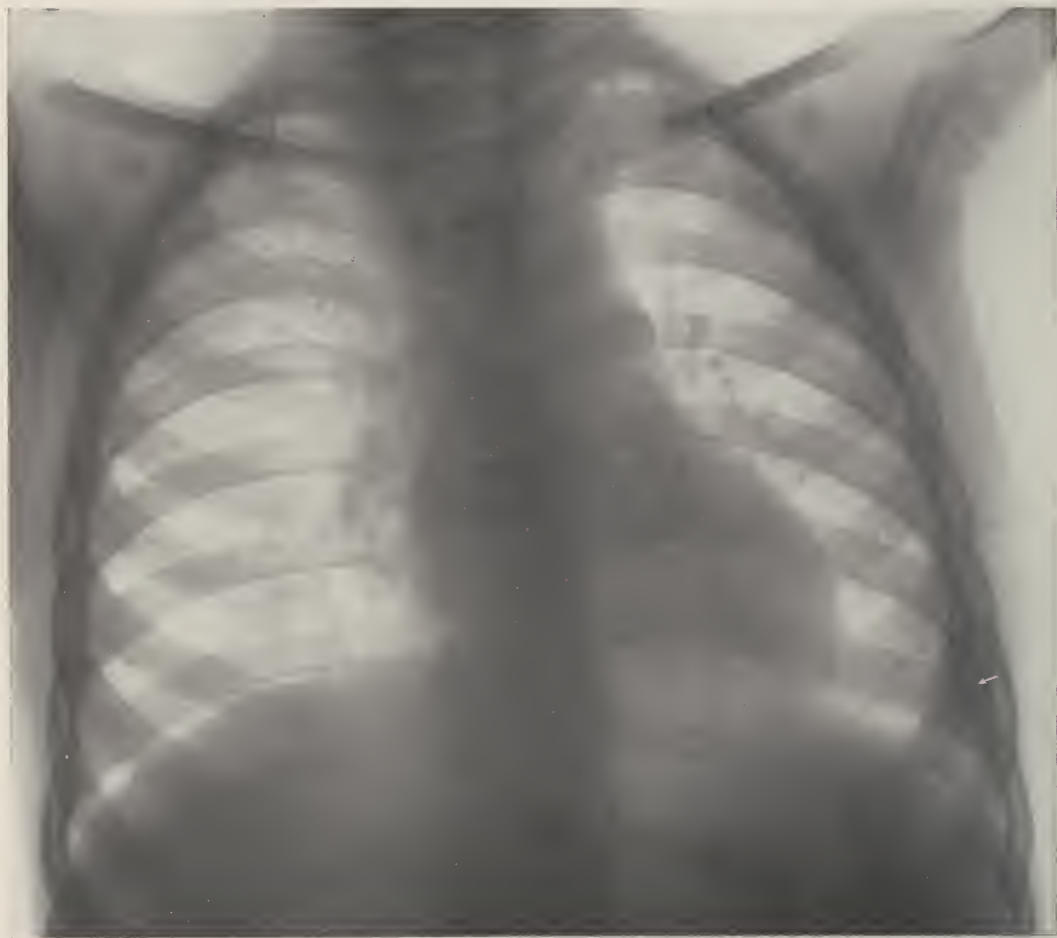


FIGURE 296

Miliary tubercles on the pleura at left base in a case of tuberculous meningitis. (arrow.)

CHAPTER XVIII

Tumors of the Pleura

The diagnosis of tumors of the pleura presents a problem of some difficulty, in the solution of which the Roentgenologist is apt to be at sea unless he has the support of clinical and pathological data. Even these aids may fail him because pleural tumors exhibit a varied symptomatology in which the pathognomonic signs of tumors found elsewhere in the body may be lacking. Thus, their remarkably long duration, extending at times over years and their clinically benign behavior, are at variance with their truly malignant character.



FIGURE 297
Endothelioma of left pleura.

In the clinical course of a pleural tumor three phases of its growth may at times be recognized. The first phase, that of initial pleural involvement is often latent. In other cases it is associated with considerable pain and for this reason it is often mistaken for simple pleurisy. The growth of the tumor soon effects a restriction of the respiratory movement and there is a rapid decrease in the volume of the affected side of the chest. On the Roentgen plate the neoplastic thickening of the pleura results in a dense, more or less homogeneous shadow which occupies the axillary zone of the chest. (Figs. 297 and 298.) In this respect they differ noticeably from primary lung tumors which originate commonly at the hilum and in which the peripheral part of the chest is only later involved. Clinically also the absence of cough and other pulmonary symptoms for a long time distinguishes pleural tumors from those developing in the lung.

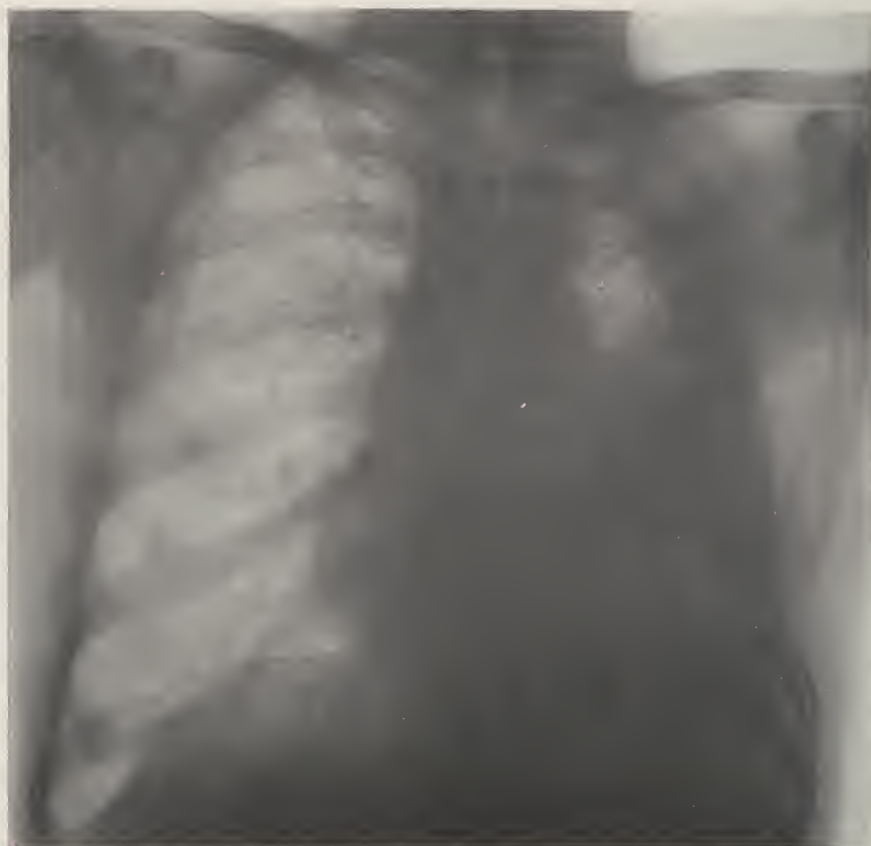


FIGURE 298

Tumor of left pleura with shrinkage of chest. Small metastases in right lung.

The growth of the tumor may early cause an agglutination of the opposing pleural surfaces so that the pleural cavity is obliterated. It is probable, however, that before this occurs, there is in most cases a pleural effusion. It is in this second phase of the disease that patients are most often observed clinically. The rapid recurrence of the fluid after evacuation, without fever or pulmonary symptoms, soon leads to a suspicion of its neoplastic origin. In these cases the Roentgen examination will do no more than reveal the pleural effusion, regarding whose nature it will give us no information, unless by chance metastases are visible in the opposite lung. The clinician here depends mostly on the microscopic examination of the pleural fluid for evidence of tumor cells.

In the third or final stage, which often succeeds that of effusion, the pleural cavity is obliterated by the growth which may be of great thickness, encasing the lung in a neoplastic shell and often invading it. Such a tumor may measurably increase the size of the lung on the affected side so that the heart and mediastinum suffer an extreme displacement to the opposite side. The sequence of events which we have described, lasting one and one-half years, beginning with

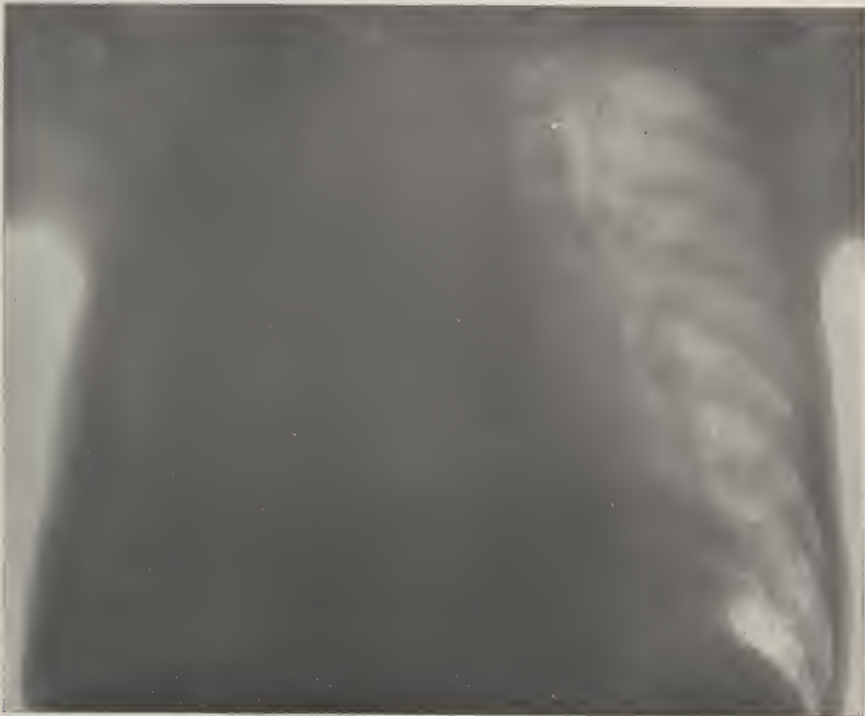


FIGURE 299

Large endothelioma of right pleura. Heart and mediastinum displaced to left. Small metastases at left base. Shadow in right chest is caused by a solid tumor of pleura of great thickness.

pleural pain and passing successively through stages of effusion and consolidation, were present in a case illustrated in figure 299. This represents the final phase of the disease in which the right pleura was completely filled with neoplasm and metastatic growths were present in the left lung. In spite of such extensive disease in the chest and metastases in the subcutaneous tissues of the flank, the patient experienced only slight dyspnoea and very little nutritional disturbance.

The following case is an instructive example of the many-sided clinical picture presented by some pleural tumors in their evolution from initial involvement to their termination:

The patient when first observed three years ago showed the symptoms and signs of a pleural effusion on the right side. Aside from the insidious onset and rather severe pain, there was nothing to indicate the neoplastic origin of this effusion. However, the finding of tumor cells in the aspirated fluid rendered the diagnosis certain. When the patient was discharged the Roentgen plate, (fig. 300,) showed an irregular and extensive thickening of the pleura. The effusion ceased and for three years the patient remained in tolerable health, having no complaint except a slight though constant pain in the right chest. There was, how-



FIGURE 300

Tumor of pleura, early stage, characterized by pleural thickening. See figure 301.

ever, a gradual, progressive shrinkage of the right chest. At her last examination, three years after the onset, the Roentgen plate, (fig. 301,) showed a marked induration of the right pleura, invasion and collapse of the right lung and a miliary involvement of the left lung. Yet clinically, so benign was this neoplasm, that in spite of this extensive process, the patient had no cough, only slight dyspnoea and was able to walk about.

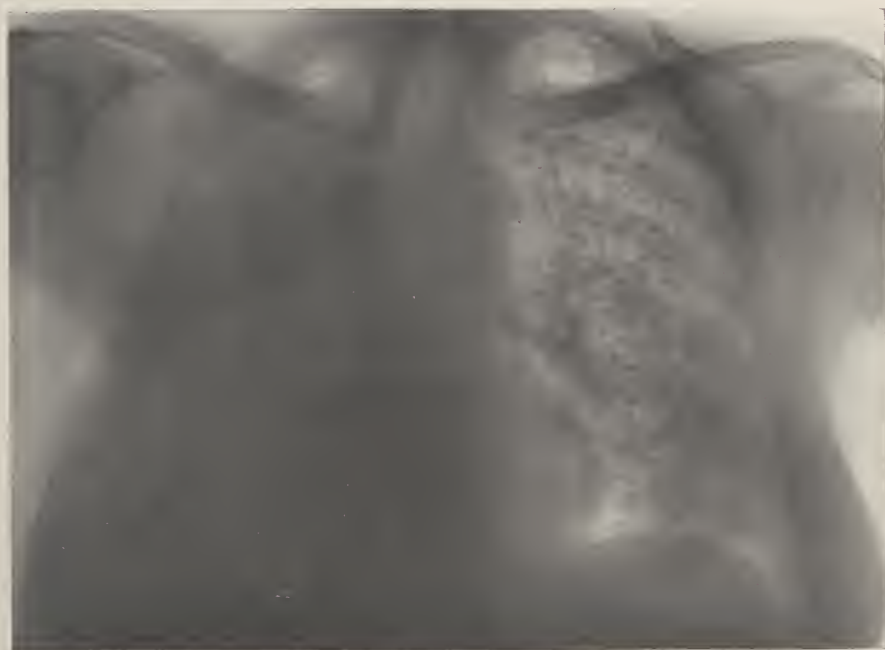


FIGURE 301

Same case as figure 300 three years later. Right chest shrunken by dense pleural growth. Miliary metastases in left lung. Ascites.

One is constantly struck by the marked contrast existing between the inordinate size of pleural tumors, especially the endotheliomata and the few symptoms which they occasion. Massive shadows which obscure nearly an entire lung field will be found in patients who, aside from slight dyspnoea, have no symptoms pointing to a serious disease. The clinical picture and the Roentgen shadows may show so little change over a period of months that the clinician may be pardoned for doubting the neoplastic nature of the disease. Yet the resistance of the tumor to the exploring needle or the decisive evidence of metastases in the cervical lymph nodes or elsewhere, usually confirms the diagnosis.

Secondary involvement of the pleura occurs frequently with primary tumors of the lungs when the latter reach the surface. On the Roentgen plate this extension rarely manifests itself by any change which would lead to its recognition. Occasionally, as in figure 302, the growth on the pleura may exceed in size the primary tumor in the lung



FIGURE 302

Tumor of pleura secondary to adeno-carcinoma of bronchus and lung.

from which it originated. In this case a small bronchial carcinoma was found in the mesial portion of the right upper lobe; the shadow on the plate, as autopsy proved, was due almost entirely to a very thick neoplastic pleura.

In conclusion, reference may be made to an interesting form of new-growth which is probably due to a subpleural lymphatic dissemination of tumor cells from the bronchial lymph nodes. When, as may happen, the region of the interlobar fissure is the site of the disease, the pleura is thickened in this situation so that it casts a linear shadow which outlines the fissure. In this so-called "fissural carcinoma" the shadow appears to extend outward from the root of the lung where it is widest, in contradistinction to the thickening of inflammatory disease in which it is widest peripherally. We have found this shadow of greatest significance in cases of carcinoma of the breast as it indicates probably metastatic disease of the bronchial nodes with retrograde involvement of the pleural lymphatics. (Fig. 303.)

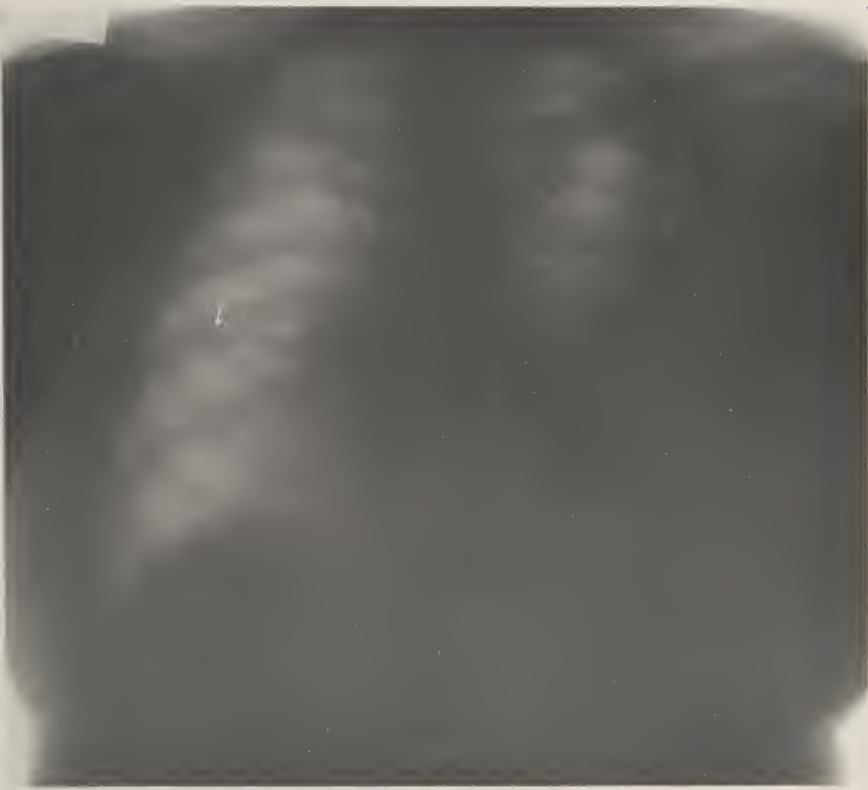


FIGURE 303

Carcinoma of pleura visible along right interlobar fissure. Secondary to carcinoma of breast. Right breast removed.

CHAPTER XIX

Chronic Pleurisy

Chronic disease of the pleura may be an isolated affection or it may be part of a pleuro-pulmonary inflammation. The latter has already been considered in the section devoted to nontuberculous disease of the lung. The effects of a chronic pleurisy may be noted on the Roentgen plate not only by the abnormal shadows cast by the thickened pleura but also by the secondary changes which operate on the other thoracic viscera.

In the first place the commonest result of a chronic pleurisy is the production of localized adhesions, whose visibility will depend on their location. Thus, diaphragmatic adhesions are most easily visible and



FIGURE 304
Unique pleural thickening following empyema.

they are rarely absent in the chronic pleurisy of pulmonary tuberculosis. Pleuro-pericardial adhesions may cause small pointed projections on the cardiac surface which are best seen in deep inspiration. A common form of chronic pleurisy is often seen in the region of the right upper interlobar fissure. It is usually a manifestation of some inflammatory process in the underlying lung, which is most often a healed tuberculosis, although it may be a sequel of acute diseases. The chronic pleurisies at the apices of the lungs which result in their retraction have already been described in the section on tuberculosis.

A slight uniform thickening of the pleura is readily missed on the Roentgen plate as it opposes no obstacle to the passage of the Ray. The



FIGURE 305

Chronic pleural thickening following absorption of pleural effusion. Note shrinkage of chest, displacement of heart and pleural adhesion at arrow.

fluoroscopic screen is better adapted to distinguish the slight difference in illumination between the two sides which such a pleural thickening will produce.

The most striking and bizarre forms of chronic pleurisy are a sequel of effusions in the pleural cavity. Especially after empyemata which have not yielded promptly to treatment, the whole parietal pleura may show an extreme induration. In other cases, a localized well demarcated area at the site of a persistent sinus, will show this change. (Fig. 304.) There is in these cases usually a marked secondary effect on the thorax, whose volume is much diminished with a consequent displacement of the heart and mediastinum. Similar changes may be found in cases of nonpurulent effusion, especially if they have been of long standing and have absorbed spontaneously. Thus in figure 305 the entire left chest is obscured and its volume is much diminished. A dense pleural adhesion extends from the apex down to the pericardium which probably represents the mesial boundary of an encapsulated effusion which had been absorbed long ago.

SECTION VI

The Mediastinum

Mediastinitis

Tumors of the Mediastinum

The Thyroid

The Thymus

Mediastinal Cysts

Aneurysm of the Aorta

CHAPTER XX

The Mediastinum

The mediastinum occupies a position in relation to the two halves of the chest which is analagous in some respects to that of the diaphragm in relation to the thoracic and abdominal viscera. Under conditions of normal intrathoracic pressure the mediastinum maintains regularly a central position. It is however, sensitive to any change of pressure on either side of it. The slightest upset in the balance of tensions exerted on it is at once registered by its displacement to the one or the other side. We have therefore in the position of the mediastinum, a delicate index of changes both in the lungs and in the pleura which the Roentgenologist is constantly on the watch to detect. This is best determined by the position of the trachea, which is readily seen on the Roentgen plate in the very middle of the upper mediastinum.

We have already, in our discussion of various pulmonary and pleural diseases, pointed out the mediastinal changes which are incidental to them. It remains for us to consider some of the diseases which originate within the mediastinum and which may affect its Roentgen shadows.

ACUTE MEDIASTINITIS

Acute inflammations of the mediastinum are usually so deeply seated that symptoms arising from them do not become apparent for some time. The mediastinum also is usually involved secondarily to organs in its vicinity, the symptoms of which commonly over-shadow the mediastinal inflammation. For this reason, it is not improbable that acute mediastinitis is often over-looked. On the Roentgen plate a diffuse cellulitis of the mediastinum will produce a general widening of its shadow which may be of help in the diagnosis, although by itself it is not characteristic.

In the recognition of mediastinal abscess the Roentgen plate may be of the greatest service because the physical signs are very difficult to interpret. We illustrate in figure 306 an example of this condition which occurred during convalescence from a lobar pneumonia. The physical signs were situated on the posterior aspect of the chest and were those of a pleural effusion encapsulated between the right lower lobe and the spine. At operation a large quantity of pus was found in the posterior mediastinum, the pleural cavity being uninvolved. The

similarity of the Rentgen picture to an effusion in the mediastinal pleura is so close, that the distinction between these two conditions can scarcely be made.



FIGURE 306

Mediastinal abscess on right side, following lobar pneumonia.

CHRONIC MEDIASTITIS

Chronic inflammatory changes in the mediastinum, whose commonest cause is pulmonary and glandular tuberculosis, occasionally make themselves manifest on the Roentgen plate. Oftenest they are evidenced by irregularities of the cardiac contour which result from adhesions between the pericardium and the mediastinal surface of the pleura. In other cases there is an actual induration of the mediastinal tissues with a consequent widening of its shadow. This is usually associated with fibrotic processes in the upper lobe. Thus in figure 307 the upper mediastinum is both widened and distorted by a chronic inflammatory process secondary to multiple bronchiectases at the root of the right lung.



FIGURE 307

Widening of superior mediastinum as a result of chronic mediastinitis, secondary to multiple bronchiectasis. Note multiple cavities at root of right lung.

In most cases these changes in the mediastinum are of no clinical importance and give rise to no symptoms. Occasionally however, the presence of mediastinal bands is responsible for disturbances in swallowing which are of sufficient severity to arouse the suspicion of organic obstruction. This is most apt to occur in cases of chronic pulmonary tuberculosis. These bands are rarely directly visible on the plate and their presence has to be inferred from the disturbed function of the oesophagus. We may thus see on the screen a momentary retardation of the ingested barium in the upper part of the oesophagus, or even a slight deviation in its course. In other cases the signs of fibrosis are so apparent in the mediastinum and the adjacent pleura and lung as to leave no doubt as to the nature of the obstruction. (Fig. 308.)

Syphilis probably ranks next in frequency to tuberculosis as a cause of chronic mediastinal disease. Unfortunately, gummatous or indurative changes are not readily seen on the plate and their presence must



FIGURE 308

Fibroid tuberculosis of left upper lobe, involving the mediastinum. Oesophageal obstruction at (x).

be inferred from such suggestive symptoms of mediastinal obstruction, as vascular engorgement of the face, a paradoxical pulse or a pleural effusion. A striking Roentgen picture is produced when the syphilitic process causes adhesions to form between the mediastinum and the diaphragm. The central tendon of the latter is then drawn obliquely along the heart border, obliterating the cardio-hepatic angle. This unique appearance is illustrated in figure 309 in which the mediastinitis is associated with a sacculated aneurysm of the aorta.

Actinomycosis is one of the rarer causes of mediastinal disease. In most cases it is probably a descending infection from the neck. The Roentgen shadow may be similar to that of a mediastinal tumor. In the case illustrated in figure 310 there were multiple sinuses in the neck from which typical actinomyces granules were obtained.



FIGURE 309

Aneurysm of aorta. Mediastinitis. Adhesion of diaphragmatic and mediastinal pleura on right side.

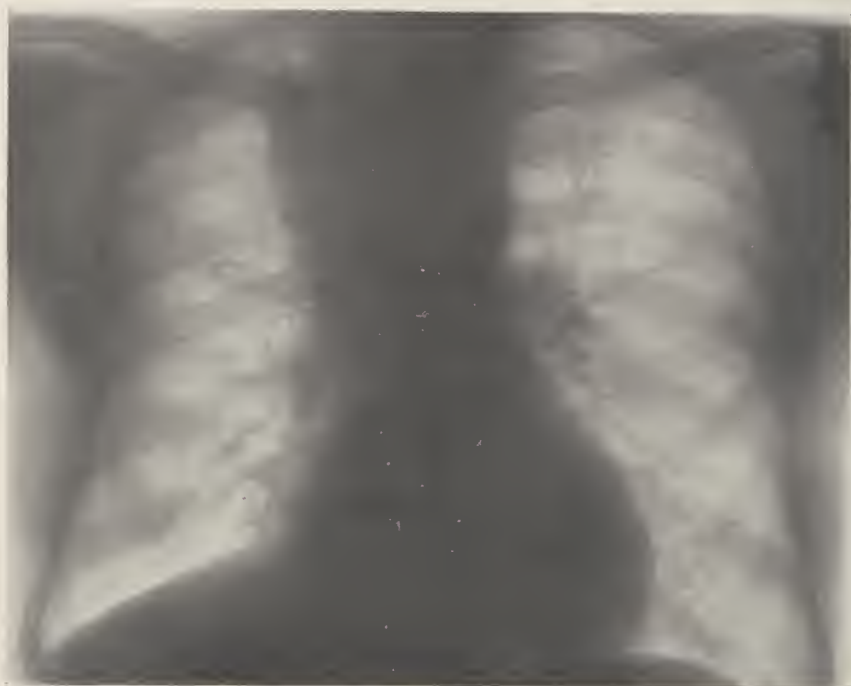


FIGURE 310

Actinomycosis of superior mediastinum. Infection extended downward from the neck.

CHAPTER XXI

Tumors of the Mediastinum

Mediastinal tumors are more often seen by the Roentgenologist than are tumors of the lung because they give rise to serious symptoms at an earlier period in their growth. For the same reason they are diagnosed earlier than are pulmonary neoplasms. The dyspnoea which is such an early and characteristic symptom of tumor of the mediastinum is apt to bring the patient quickly to his physician; on the other hand the cough and expectoration which mark the onset of a tumor of the lungs are such common symptoms of less serious respiratory diseases, that the patient is prone to disregard them for a considerable time.

From the Roentgen standpoint, mediastinal tumors are best classified as to their location. We can distinguish in the first place those which involve the superior mediastinum from others which develop in its lower portion or finally from those which completely fill it from apex to base. Their frequent location in the upper mediastinum can be accounted for by the presence in this region of the bronchial lymph nodes, from which a large number arise and also by the presence there of the thyroid the thymus and epithelial inclusions which may be the occasional sources of mediastinal tumors.

The shadows cast by mediastinal tumors are homogeneous and dense. They stand out sharply against the surrounding lung and only rarely show evidence of infiltration of the latter, although this is often seen at autopsy. They are nearly always bilateral, especially when they are well grown and this in most cases will serve to distinguish them from primary tumors of the lung. The growth on the two sides is however unequal in extent. Fluoroscopic examination in the oblique positions may be of considerable help in determining whether the growth originates in the lung or the mediastinum.

The notable result of the growth of a mediastinal tumor is an increase in the width of the median shadow which projects for a variable distance into the lung. This shadow may exhibit the greatest diversity both in size and shape, depending on the situation and the mode of growth of the tumor. Thus, the outlines of the mass may be smooth and lobulated or irregular and infiltrating, or even rectilinear. Its greatest length may be vertical or again it may extend transversely across the chest. In the latter case it may be difficult to distinguish it from a hilum carcinoma.

The smaller tumors, which barely project from the root of the lung, as is to be expected, will present little that is characteristic upon which to base a diagnosis of such a serious disease. It will thus not be easy to differentiate the moderate increase in size and density of the root shadows from that due to indurated lymph nodes. The diagnosis will here be suggested by such unusual symptoms as deep thoracic pain or a recurrent laryngeal paralysis which are more readily explained by the assumption of a tumor than otherwise. Thus, in figure 311 the small lobulated tumor at the left root produced only a paralysis of the left vocal cord and considerable pain, without other symptoms or physical signs.



FIGURE 311

Beginning mediastinal tumor casting lobulated shadow at left root. Symptoms were precordial pain and paralysis of left vocal cord.

The larger mediastinal sarcomata produce such characteristic Roentgen appearances as to leave no doubt of the diagnosis. We may illustrate them, in their great variety, by the following cases. In figure 312 is shown a large bilateral tumor which involves the greater part of the mediastinum, the heart being encased in it. This was apparently a slowly growing tumor; it is remarkable that such an enormous mass occasioned few symptoms and caused no embarrassment of the heart's



FIGURE 312

Large mediastinal tumor encasing the heart.

action. In contrast to this, a rapidly growing tumor, although much smaller in size, may produce extreme dyspnoea and cyanosis by compression of the trachea and bronchi. The mediastinal sarcoma shown in figure 313 had existed for only four weeks yet the patient was at the point of death from asphyxia. The semi-circular shadow at the right of the heart represents probably a rapidly growing portion of the tumor. The patient presented all the clinical signs of a compression of the vena cava.



FIGURE 313

Mediastinal tumor. Rapidly growing lympho-sarcoma. (arrow.)

The lobulated border of mediastinal tumors is their most constant Roentgen feature. This is clearly indicated in figure 314, a case of angio-sarcoma which produced such a degree of dyspnoea that a thoracic decompression was required. The exaggerated mottling of the pulmonary fields which was due to venous engorgement, is a striking evidence of the pressure of this tumor on the roots of the lungs. Lobulation is also manifest in the tumors shown in figures 320 and 325. All of these were lympho-sarcomata.

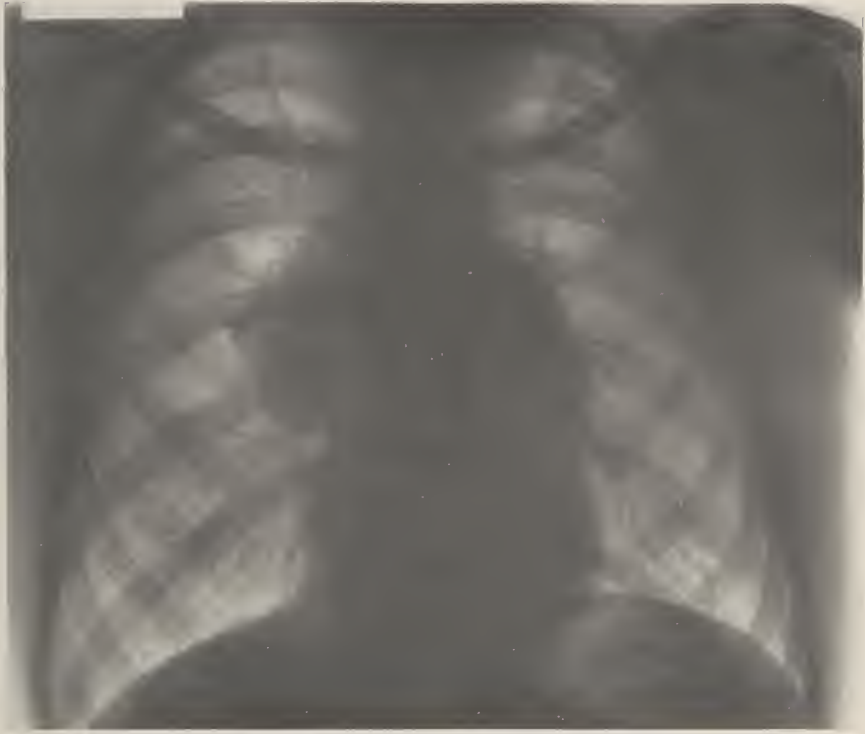


FIGURE 314
Mediastinal tumor. Angio-sarcoma.

In the rare, infiltrating new-growths the smooth lobulated contour is at times lost. Their outlines may be indistinct and they may shade off into the surrounding lung. Thus in figure 315 the infiltrating mass, which at autopsy proved to be a lympho-sarcoma, extends transversely across the chest, very much like a carcinoma. Again, in figure 316, the ray-like projections from the tumor may be regarded as an infiltration of the pulmonary tissues. In figure 317 the mediastinal growth extensively invades the right upper lobe.

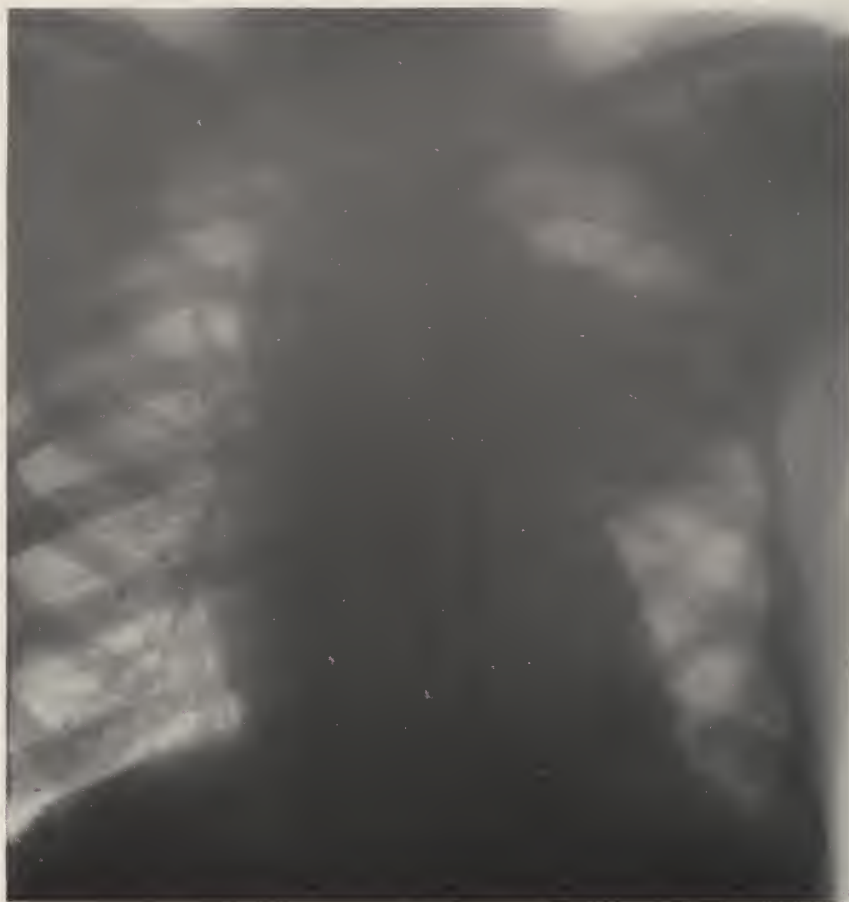


FIGURE 315

Mediastinal tumor. Lympho-sarcoma. Associated with cervical and axillary Hodgkin's disease.



FIGURE 316
Mediastinal tumor. Infiltrating type.

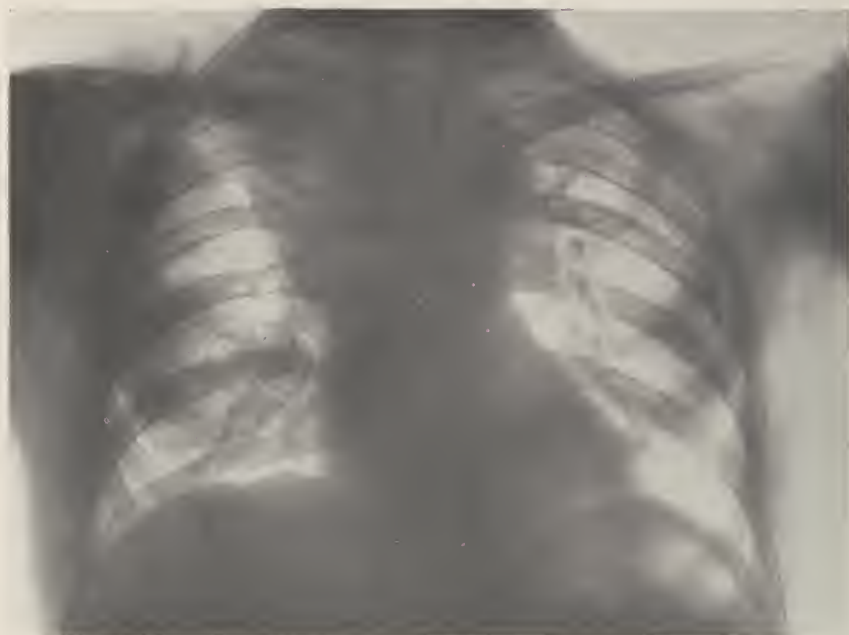


FIGURE 317

Mediastinal tumor invading right upper lobe.

The distinction between carcinoma and sarcoma of the mediastinum cannot be made from the Roentgen examination. Carcinoma is undoubtedly rare and for this reason only some unusual circumstance would make the diagnosis a safe one. Aside from metastatic carcinoma of the bronchial lymph nodes, which rarely is responsible for the clinical picture of mediastinal tumor, we have observed only two cases of carcinoma of the mediastinum. They were both unilateral tumors and exhibited the invasive tendencies of cancer by a perforation of the oesophagus. The unusual character of the cases may be illustrated by the following history:

A man of sixty years, for some months had the symptoms of a new-growth at the cardia. He had great difficulty in swallowing and was much emaciated. One day before his death he vomited a large amount of necrotic material. At autopsy there was found, in the posterior mediastinum, immediately behind the heart, an encapsulated, broken down tumor about four inches in diameter, which had ulcerated into the lower end of the oesophagus and in this manner discharged its contents. Microscopic examination proved this tumor to be an epithelial new growth, which developed probably from fetal rests.

The plate, (fig. 318,) shows the outline of this tumor, which is almost obscured by the heart shadow, projecting slightly beyond its right border. The main mass of the tumor however, lay behind the heart and for this reason the interpretation of the shadow was uncertain.



FIGURE 318

Tumor of posterior mediastinum. Necrotic carcinoma which perforated the lower oesophagus. Tumor partly concealed by the right side of heart. (arrow.)

Secondary mediastinal tumors are more often due to carcinoma than to sarcoma. Ordinarily the new-growths, consisting of cancerous lymph nodes, remain small and do not attain the size of a tumor mass. On the other hand, in occasional cases these nodes may become so large that they completely overshadow a primary neoplasm of the lung from which they originated and whose existence is unsuspected. The mediastinal shadow seen in figure 319 presented the typical appearance of a primary mediastinal tumor, which caused marked dyspnoea from pressure on the trachea and bronchi. The bronchoscopic examination was in harmony with this supposition as it revealed a narrowing of the trachea at the bifurcation, by an extrabronchial mass. At autopsy, the tracheo-bronchial nodes were enormously enlarged and cancerous; the primary tumor however, was very small and was of the rare infiltrating type of carcinoma of the wall of the bronchus which did not invade the lung. The Roentgen shadows were entirely due to the metastatic cancer of the bronchial nodes. The significance of the initial symp-



FIGURE 319

Metastatic carcinoma of mediastinal and bronchial lymph nodes. Primary tumor in bronchus was latent. Clinical picture of heart failure. Note vascular engorgement of lungs. Effusion at right base.

toms in this case, which were obstinate cough and hemoptysis was only realized when the primary bronchial origin of the tumor was determined at autopsy.

A study of the plates in cases of mediastinal tumor often throws light on the origin of the distressing symptoms which are occasioned by the pressure of the growth on neighboring structures. Owing to their frequent occurrence in the upper, narrow portion of the chest they are prone to compress and displace the trachea, bronchi and oesophagus, the symptoms of which are the immediate occasion of the patient's visit to the physician. On the plate, the narrowing and displacement of the trachea are readily seen. A similar involvement of a main bronchus is to be inferred from an atelectasis of a lobe and from the other fluoroscopic evidences of broncho-stenosis which have already

been described. The position of the heart is not ordinarily influenced by a mediastinal growth because the latter is usually bilateral and because the bulk of the tumor is commonly situated in the upper mediastinum away from the heart. Here it may depress the arch of the aorta or displace it laterally. Only when the growth is disproportionately large on one side or when there is an associated pleural effusion, may the heart suffer a notable displacement. (Figs. 320 and 321.)

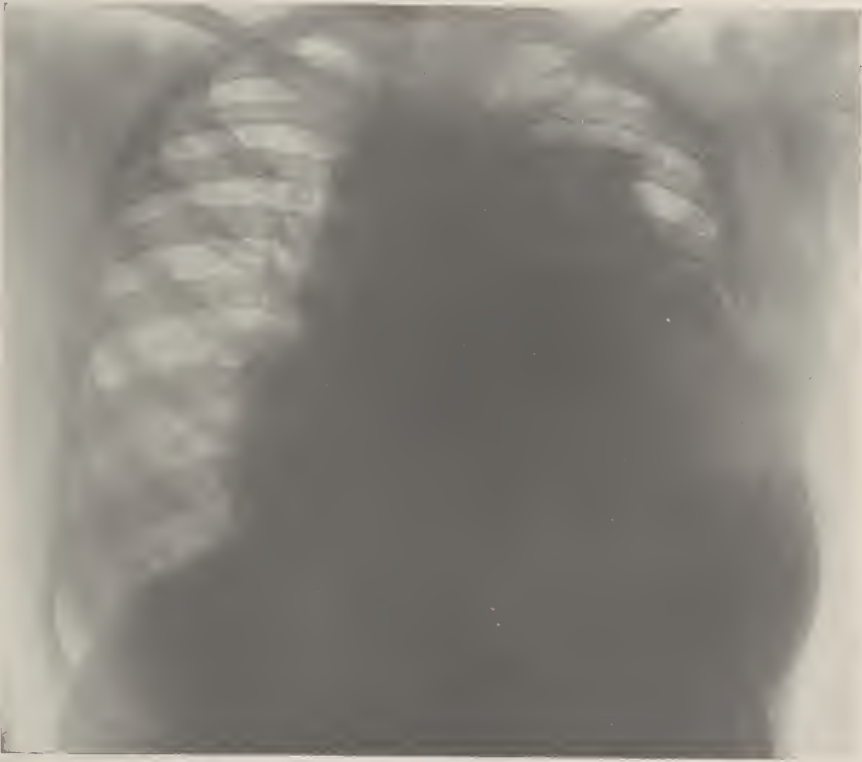


FIGURE 320

Mediastinal tumor with lobulated border. Heart displaced. Pleural effusion.

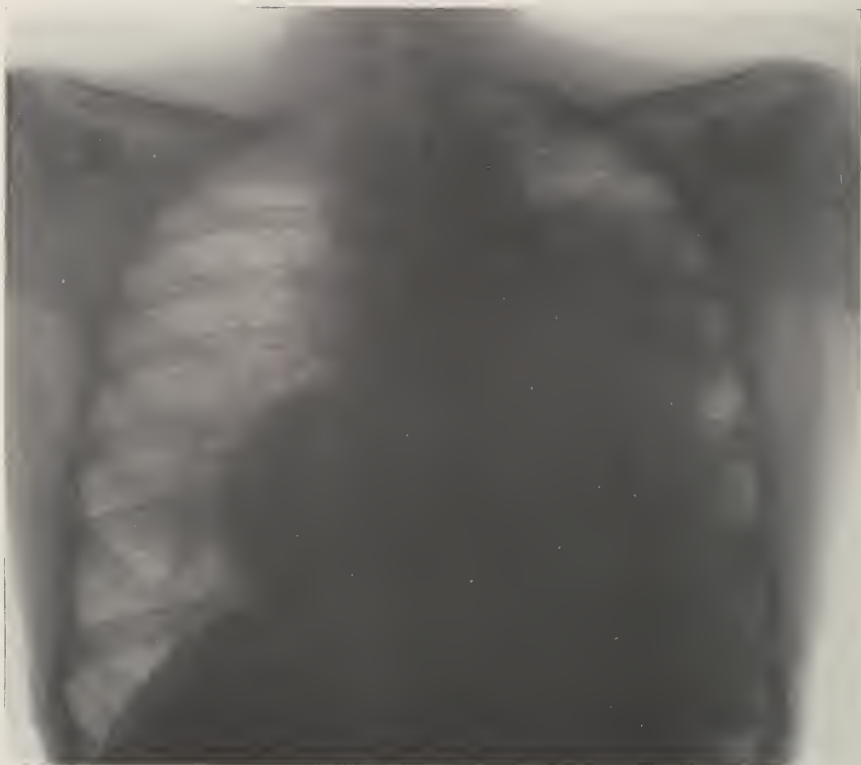


FIGURE 321

Mediastinal tumor. Heart displaced to the right.

COMPLICATIONS

One of the most frequent results of a mediastinal tumor is a pleural effusion, which is usually unilateral. It is found in the terminal stage in most cases and is then probably due to venous compression. Thus, in figure 322 the massive lymphomata at the root of the lung were responsible for the effusion which half filled the right chest. In other cases, the effusion may be a relatively early symptom of the disease and it may be interpreted as an extension of the growth to the pleural membrane which is actually invaded by the tumor. The sequence of the symptoms indicating pleural involvement may be significant, especially the onset of pleural pain, which is unusual with mediastinal growths.

For example, a woman of nineteen years, complained for three months of dyspnoea, cough and edema of the legs. Latterly she suffered in addition from severe pains in her left chest. On physical examination her chest was prominent and contained a large quantity of bloody fluid which entirely obscured the underlying tumor. After removal of the latter a large lobulated mass was seen extending from the left mediastinum, which displaced the heart to the right. (Fig. 320.) The pleural fluid contained large numbers of tumor cells which presumably were derived from the pleura.

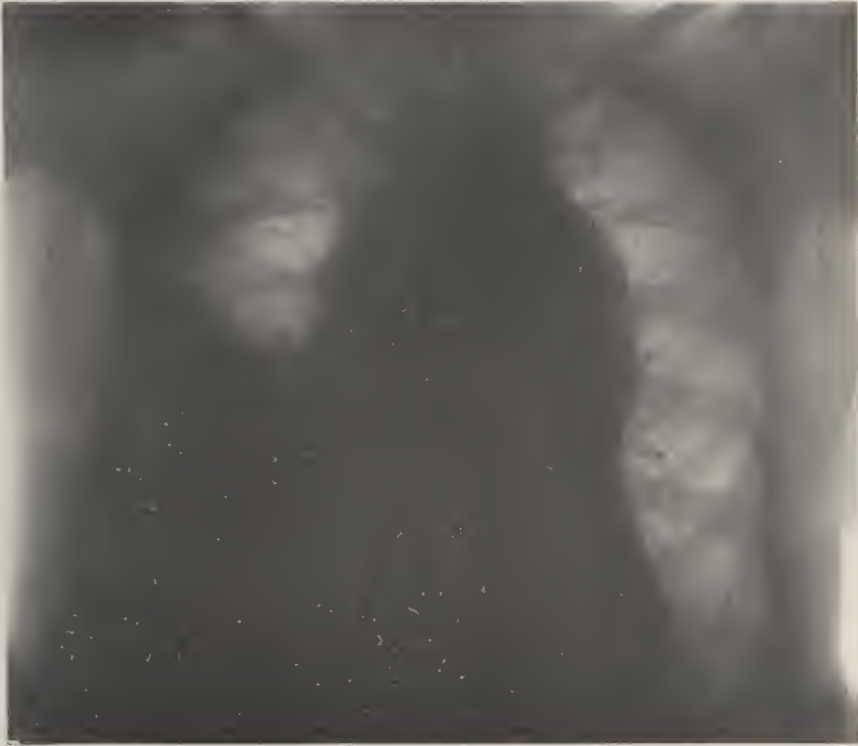


FIGURE 322

Mediastinal tumor. Chronic leukemia. Pleural effusion.

In rare cases, very large effusions may be the result of pressure on the root of the lungs, by tumors which are either obscured by the effusions, or which are so small that they are completely hidden by the heart. One is here reminded of the metastatic carcinomata in the bronchial lymph nodes following cancer of the breast. The characteristic feature of these cases is a persistent dyspnoea with an effusion which rapidly re-accumulates after its evacuation. The Roentgen examination in these cases does not clear up the diagnosis and dependence must be placed on examination of the pleural fluid for evidence of blood or tumor cells.

Aside from their direct involvement by extension of the tumor into them, the lungs may show true metastases which are similar in appearance to those following new growths elsewhere in the body. (Fig. 323.) The Roentgen plate may also show abnormal shadows due to metastases in other thoracic structures such as the thyroid.



FIGURE 323

Mediastinal tumor with metastases in the lungs.

The occasional compression of the phrenic nerve by mediastinal tumors is reflected on the fluoroscopic screen by a high paralytic diaphragm on one side. This must not be confused with the high diaphragm occasionally seen with chronic mediastinitis, which may complicate both mediastinal tumors and aneurysms. (Fig. 309.)

DIFFERENTIAL DIAGNOSIS

The shadows cast by mediastinal tumors are so characteristic that the necessity of distinguishing them from other intrathoracic diseases rarely arises. They are more often confused with aneurysms of the aorta than with any other condition. (Fig. 324.) The resemblance is at times so close that only the growth of the tumor as determined by a subsequent Roentgen examination will clear up the diagnosis. (Fig. 325.)

A mediastinal tumor may be so situated as to simulate an enlargement of some portion of the heart. It may thus resemble an enlargement of the right auricle, as in figure 318. In figure 326 the small mediastinal



FIGURE 324

Mediastinal sarcoma resembling aneurysm of the aorta.



Mediastinal lympho-sarcoma showing growth of tumor after four months.

FIGURE 325



FIGURE 326

Mediastinal tumor. Acute myeloblastic leukemia. Simulates enlargement of pulmonary artery.

tumor has the position and shape of a dilated pulmonary artery. In this case, no confusion in diagnosis arose, because there was an absence of pulsation, whereas an enlarged pulmonary artery practically always exhibits a marked pulsation.

The introduction of gas into the pleural cavity may be of decided service in distinguishing mediastinal growths from other diseases, especially encapsulated effusions, which may at times cast the dense circumscribed shadow of a tumor. For example, the shadow in the right upper chest in figure 327 had all the characteristics of a localized effusion. After the introduction of air into the chest and the collapse of the lung, it was at once apparent that a solid tumor was present. This was found at operation to be a ganglio-neuroma.



FIGURE 327

Mediastinal tumor. Ganglio-neuroma. Artificial pneumothorax with collapse of lung served to distinguish this tumor from an encapsulated effusion.

ROENTGEN-CLINICAL CORRELATIONS

The more general use of the Roentgen Ray will lead, it is hoped, to an earlier diagnosis of obscure intrathoracic diseases such as tumors of the lungs and mediastinum. This is desirable, among other reasons, because the symptoms which usher in a mediastinal tumor may be so little characteristic that they are often mistaken for those of pulmonary tuberculosis or even of trivial affections such as bronchitis. Physical signs are rarely present at this early stage of the disease and it is not until major and urgent symptoms make their appearance that the seriousness of the situation is appreciated. Although it is too much to hope that all patients who present such minor symptoms can be radiographed, there are certain symptoms the presence of which should make a Roentgen examination imperative. The significance of laryngeal paralysis, for example, as an evidence of new growth of the mediastinum cannot be overestimated. This symptom should always lead to a search for some mass in the superior mediastinum, which is often rewarded by the discovery of a tumor or an aneurysm. See figure 311.

A rapidly developing and persistent oedema of one arm will in the same way suggest the likelihood of a mass in the upper mediastinum which is causing pressure on the subclavian vein with thrombosis. In such cases the plate will often repay the examiner for his efforts. Thus, the mediastinal shadow in figure 342 was found at autopsy to be due to a tumor of the thymus. The pressure of this tumor on the left subclavian vein caused oedema of the arm, which was almost the first symptom of the tumor growth.

In addition to these more obvious signs which point to mechanical pressure, it is well to bear in mind that mediastinal tumors, especially while they are yet small and undiscovered, may produce symptoms which are easily confused with those arising in neighboring organs. Symptom complexes due to intrathoracic pressure may thus arise which will bear a striking resemblance to various forms of heart disease. An initial symptom of mediastinal tumor may be a deep seated pain in the chest which may be so like that of angina pectoris as to justify the diagnosis of coronary disease. This error was made in a patient whose only symptom for some time was the typical precordial pain of angina pectoris; the subsequent development of a recurrent paralysis led to the Roentgen examination which revealed a small tumor at the root of the left lung. (Fig. 311.) There was neither cough nor dyspnoea.

In another form of tumor, whose growth is manifested especially by a stenosis of the trachea or bronchi the clinical picture of decompensated heart disease may be simulated to the smallest detail. The dyspnoea, cyanosis, tachycardia, pleural effusion and even a cardiac murmur would seem to leave no room for doubt as to the cardiac origin of the symptoms. It will be noted however that neither the evacuation of the pleural effusion nor the administration of digitalis brings relief to the patient and the marked engorgement of the veins of the face and neck will suggest a mediastinal obstruction. In the case illustrated in figure 319 in which these symptoms were present, the discovery of the upper mediastinal tumor which compressed both main bronchi supplied an adequate cause for them outside of the heart. The Roentgen appearance of the heart, which was little enlarged, was not such as would be expected in a decompensated valvular lesion capable of producing such alarming symptoms. The tumor was found at autopsy to be a mediastinal carcinoma secondary to a bronchial neoplasm.

TUMORS OF THE PERICARDIUM

New growths of the pericardium are of extremely rare occurrence and the difficulty in their diagnosis is proportionate to their rarity. It is desirable therefore whenever the opportunity presents itself to make known the findings in cases in which the diagnosis is proven. The large shadow in figure 328 which is co-extensive with that of the heart was



FIGURE 328
Sarcoma of pericardium.

found at autopsy to be due to a spindle-cell sarcoma which formed a layer, in places one inch thick, about the heart, completely obliterating the pericardial cavity. In places the tumor invaded the heart muscle. During life the symptoms were those of a pericardial effusion which produced considerable cardiac embarrassment. Aspiration of the pericardium revealed a small amount of bloody fluid in which some atypical cells, suggesting tumor cells, were found. The Roentgen plate by itself would not warrant the diagnosis of such a rare condition; the heart silhouette is that found in cases of an asymmetrical distension of the pericardium with fluid.

CHAPTER XXII

The Thyroid Gland

In the diagnosis of disease of the normally situated thyroid gland the Roentgen Ray occupies a position of minor importance. A cervical enlargement of the gland can more easily be determined by palpation and the exact pathological change in it can readily be ascertained. The normal thyroid is usually not visible on the plate because its shadow, which is of only slight density is obscured by the spine and the muscles of the neck. Slight degrees of hyperplasia of the gland are however so common, especially in women, that it is not unusual to see ill-defined shadows of the thyroid at the root of the neck which extend a short distance beyond the margin of the sterno-mastoid muscles into the apical pulmonary fields. A similar shadow is often seen in cases of exophthalmic goiter.



FIGURE 329

Small substernal goiter on right side. Cervical thyroid of normal size.

When the shadow of a cervical thyroid is at all dense the pathological change has usually gone beyond simple hyperplasia; in such cases colloid or cystic goiters will be found, often associated with a visible compression of the trachea. (Fig. 330.)

The Roentgen examination plays a greater role in the diagnosis of substernal or intrathoracic enlargements of the thyroid. By means of it a downward extension of thyroid tumors may be visualized and their relation to adjacent structures defined. Goitres are in only rare instances entirely intrathoracic. Nearly always the intrathoracic portion is a downward prolongation of a cervical goiter which extends in some cases only as far as the clavicle or the manubrium and in others further invades the chest down to the heart. Roentgen study of cases of colloid and cystic goiters has shown that such a downward extension of a cervical struma occurs in a large percentage and that little dependence can be placed on the physical signs to determine its downward growth.

The intrathoracic portion of an enlarged thyroid is rarely symmetrical; usually one side is larger than the other, this being more often the right side. The thyroid shadow is a uniformly dense one and it is



FIGURE 330

Cervical goitre extending a short distance into chest.

continuous with that of the aorta which it often overlies. Its borders are smooth, convex and sharply defined.

The Roentgen features of intrathoracic thyroids are brought out in the following cases. In figure 329 the unilateral thyroid tumor is entirely substernal, the cervical portion of the gland being normal in size. This goitre is of a type not uncommonly seen; in spite of its small size and the absence of physical signs it may be the cause of pronounced pressure symptoms. More often the growth is bilateral and it extends a greater distance into the chest. In figure 330 the cervical colloid goiter just dips into the upper aperture of the chest; in figures 331 and 332 the downward growth is considerable.



FIGURE 331

Symmetrical enlargement of an intrathoracic goiter. Calcification in right lobe of goiter. Trachea displaced to left.



FIGURE 332

Substernal thyroid. Displacement of the trachea to left side.

As a rule the pathological type of the goiter cannot be inferred from the appearance of the Roentgen shadows. It is probable however, that the denser shadows are associated with the presence of much colloid whereas the fainter ones represent adenomatous changes.

There is no essential difference between the shadow of a benign and a malignant tumor of the thyroid. The latter shows no infiltrating border and it remains well circumscribed throughout its course. It therefore happens that when carcinoma develops in a previously existing goiter, aside from an increase in its size, there may be nothing to indicate that the pathological process has changed. For example, the globular tumor situated behind the sternum in figure 333 was found at operation to be a carcinoma of the isthmus. This tumor had existed, according to the patient, for fifteen years with only minor symptoms. The Roentgen shadow afforded no basis for a diagnosis of malignancy and it differed in no wise from a cystic enlargement of the gland. Only the sudden onset of severe dyspnoea aroused a suspicion of cancer. In this connection it is to be borne in mind that benign growths of the thyroid may also take on a rapid growth with urgent dyspnoea. Here again the Roentgen ray is likely to fail us in the distinction between a benign and a malignant struma.

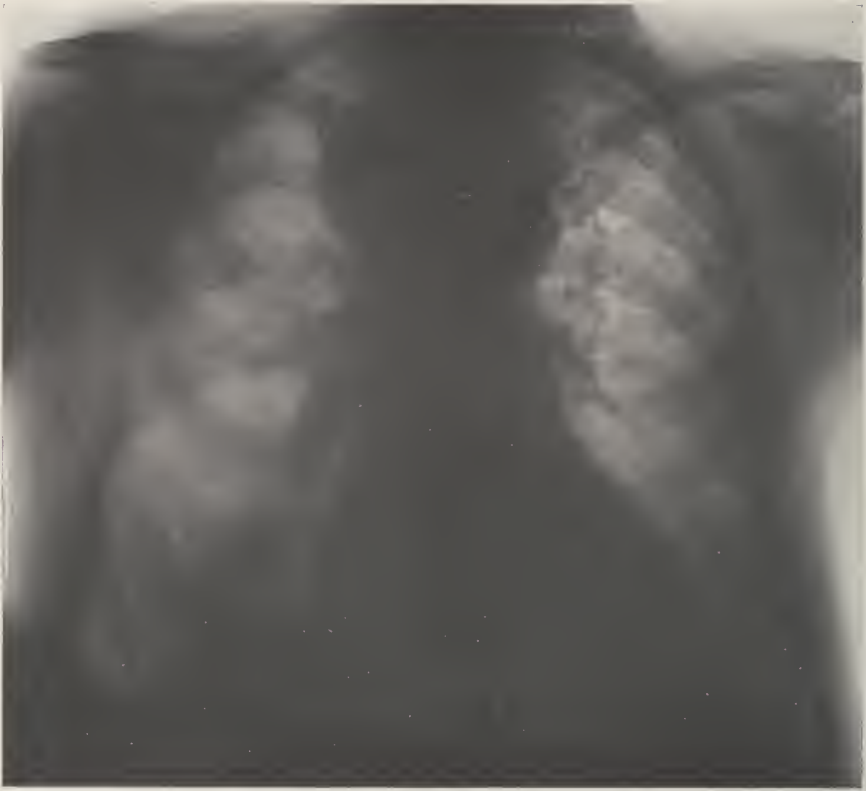


FIGURE 333

Adenocarcinoma of the isthmus, developing in a goiter of 15 years' standing.

In order to determine the malignancy of a goiter on the Rentgen plate we must therefore look elsewhere than in the thyroid shadow itself. The presence of metastases in the lungs may be the first sign of a cancerous degeneration of the goiter. For example, in figure 225 the purely cervical goiter, whose shadow on the plate appeared innocent enough, gave rise to numerous metastases in the lungs.

Bearing in mind the frequency with which cancers of the thyroid metastasize in the bones, the Roentgenologist will not fail to make a close inspection of the bones of the thorax in order to discover secondary tumors in the spine or in the ribs which may have resulted in pathological fractures. In fact, pain in the bones of the thorax or elsewhere in the body may be the first clinical evidence of malignant disease of the thyroid and it is a peculiarity of neoplasms of this gland that the primary tumor may be very small and yet give rise to large secondary tumors.

When we investigate on the plate the relation of the enlarged thyroid to the other structures in the upper part of the chest, we find graphically portrayed the reason for its most striking symptom namely, dyspnoea. With the larger tumors the trachea suffers varying degrees of compression and, depending on the conformation of the growth, the trachea is displaced either to the right or to the left. (Figs. 332 and 334.) Even with minor grades of thyroid enlargement, whether this be



FIGURE 334

Unilateral intrathoracic goiter of rapid growth. Marked displacement and compression of trachea indicated by arrow.

in the neck or within the thorax, the trachea rarely escapes some degree of constriction. So true is this that whenever on the Roentgen plate the course of the trachea is altered or its caliber narrowed, a thyroid enlargement may be suspected, even though no definite shadow of the gland can be seen. The only other common cause of such a change in the trachea is a chronic tuberculous process in the lungs, which is of course, easily recognized.

The greatest compression occurs at the superior aperture of the thorax, that is, at its narrowest point; here the trachea may have its calibre reduced to that of a lead pencil. Yet, smaller growths, if they are favorably placed may exert an even greater degree of pressure than the larger ones. Thus the tumor of the isthmus in figure 333 is relatively small in size, yet it is so situated that the trachea, as it traverses the upper chest, is thrust from its normal course and compressed between it and the arch of the aorta.

Other pressure effects, such as a downward displacement of the aortic arch, can occasionally be seen on the plate. Noncancerous goitres rarely come into relation with the oesophagus and are therefore not prone to cause dysphagia. Carcinomata on the other hand may invade it and thus interfere with deglutition. When the thyroid tumor is large, the cause of the symptoms is at once obvious. Occasionally the new growth is so small and its Roentgen shadow so vague that visualization of the oesophagus with barium may be necessary in order to establish the site of the constriction and its relation to the thyroid.

The cardio-vascular symptoms associated with goitres are of more than ordinary interest. Aside from the more obvious circulatory disturbances, such as cyanosis and engorgement of the superficial veins of the neck and chest which result directly from the pressure of the large

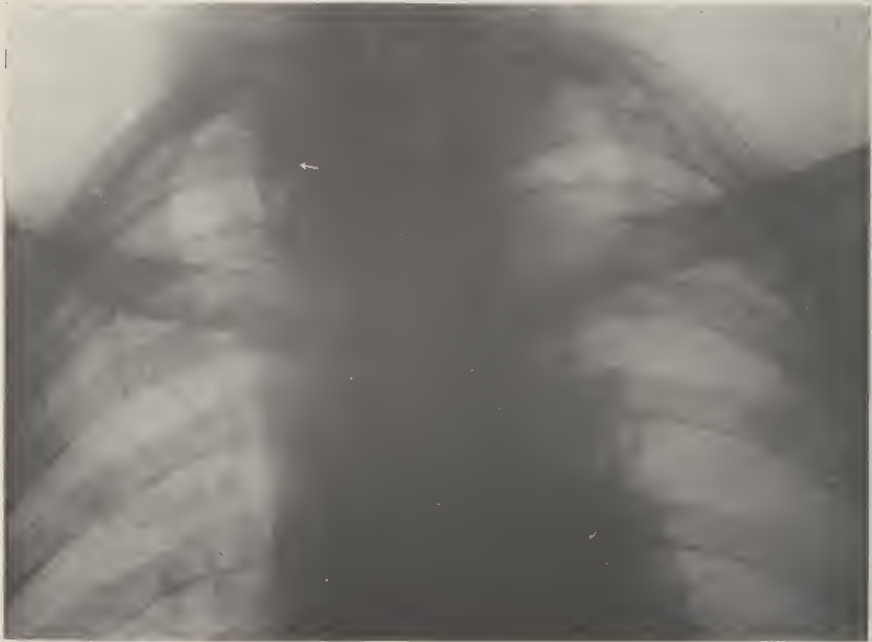


FIGURE 335

Lime deposits in right lobe of thyroid.

thyroid, our interest is arrested by an obscure group of cases in which a marked derangement of cardiac function seems to stand in some relation to a substernal thyroid. In general, these patients present the picture of myocardial disease, the striking clinical manifestation of which consists in a fibrillation of the auricle. The heart often shows an enormous enlargement for which no adequate cause can be found in the previous history nor in the present status of the patient. If we examine the superior mediastinum in these cases a substernal thyroid will not infrequently be found. The shadow of the latter will oftenest be seen on the right side beneath the inner extremity of the clavicle; owing to its small size and the faintness of the shadow, it is better seen on the screen than on the plate. This shadow must of course, not be confused with the innominate vessels which are occasionally prominent in this situation. Whether we are warranted in assuming a relation between the cardiac disease and the enlarged substernal thyroid may well be open to doubt. None the less the clinician cannot fail to take a lively interest in the discovery of pathological changes in the thyroid in cases of circulatory disease whose causation is obscure and he will seek to ascertain by appropriate tests of thyroid function what relation, if any, exists between the heart condition and the thyroid enlargement.

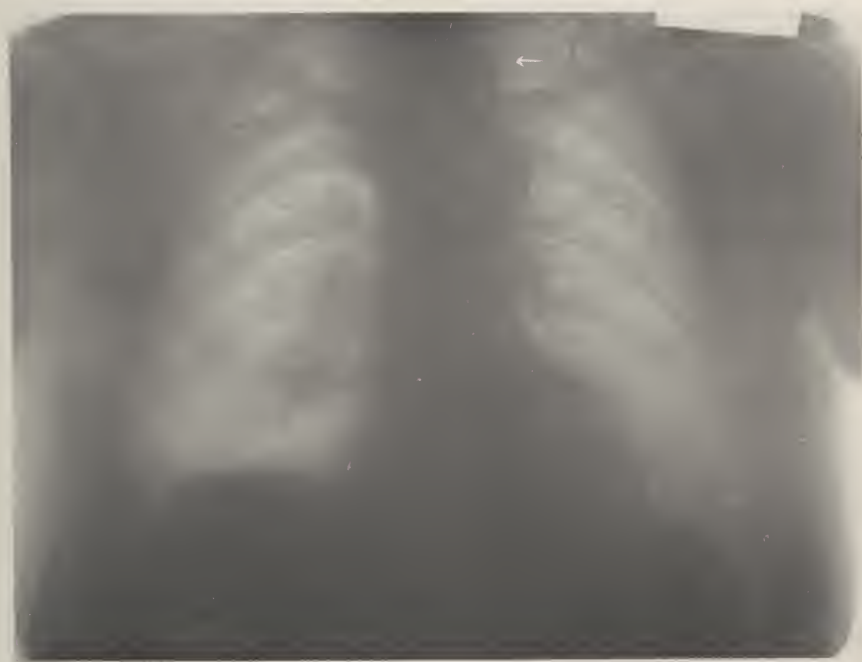


FIGURE 336

Calcification of an old colloid goiter of the isthmus. (See figure 337.)

In addition to an enlargement of the thyroid, the Roentgen plate at times reveals more minute changes in it. Of these the most striking is a calcification of the gland which may be seen in one of the lobes, as in figure 335, or in an enlarged isthmus. (Figs. 336 and 337.) In the



FIGURE 337

Oblique view of figure 336, showing calcification of the thyroid isthmus.

latter case the unique arrangement of the lime deposits is best brought out in the oblique position. These concretions are usually seen in long standing goitres, with colloid degeneration. (Fig. 331.)

CHAPTER XXIII

The Thymus

At birth, the thymus is relatively large and occupies an appreciable space in the superior mediastinum. It is however so situated between the sternum in front and the spine behind that its Roentgen shadow is obscured by these bones. Laterally its borders do not extend beyond the vena cava and aorta which normally limit the mediastinal shadow. Below it merges with the heart and the aorta from which it is not distinguishable.

An enlargement of the thymus will make itself manifest on the plate by a widening of the superior mediastinum and also by a change in its contour. As this condition has a clinical importance especially in infants and young children, it is necessary to familiarize ourselves with the appearance of the mediastinum in them in order that we may recognize deviations from the normal. If, with this object in view, we examine the chests of presumably normal infants a surprising variability in the width of the mediastinum will be found. In some cases, it is so narrow as to lie entirely behind the shadow of the sternum whereas in others it may extend laterally to the parasternal line. This variability in the width of the mediastinum is little, if at all, dependent on the size of the thymus. It is rather due to a change in the volume of the vascular structures contained within the mediastinum, brought about by extraneous causes such as the posture and respiration of the child, whereby the large vessels, especially the vena cava, are more or less distended with blood. Thus, when the child assumes the prone position in which the exposure is usually made, these vessels, relieved of the weight of the heart, become shorter and broader and their shadow is correspondingly altered. An even greater influence on the mediastinal shadow is exerted by the respiratory movements. During deep inspiration the vena cava, which marks the limits of the mediastinum on the right side, is emptied of its blood so that its shadow approaches the mid-line. The mediastinum is therefore narrowed. During expiration or when the breath is held, as it frequently is when the child cries, the vena cava is distended with blood and the mediastinal shadow is widened. So marked may this change be that two plates made in different phases of respiration may be so unlike that they will scarcely be recognized as belonging to the same child. (Fig. 338.)

Of equal importance in estimating the width of the mediastinum is the position of the patient with respect to the tube. The latter must be centered exactly over the mid-line of the chest. The slightest rota-



FIGURE 338

Appearance of the mediastinum in deep inspiration. Note low position of diaphragm.

Shows widening of mediastinum in same infant during expiration. Note high position of diaphragm.

tion of the chest will also entirely vitiate the examination as it will cause a broadening of the mediastinal shadow and project it outward into one or the other pulmonary field.

It must be evident from the preceding observations that the mediastinal shadow in children is not a fixed, unalterable one. Its interpretation is fraught with difficulties and a widening can only be regarded as evidence of disease, when the above mentioned sources of error are eliminated. There is no doubt that many so-called cases of thymic enlargement have been reported in the literature, in which the diagnosis is open to serious doubt because of failure to observe these necessary precautions in the Roentgen interpretation of the normal mediastinum.

The Roentgen examination for an enlarged thymus is usually undertaken in infants to explain the stridor and dyspnoea which characterize attacks of thymic asthma. In newborn infants particularly, such attacks will at once arouse a presumption of enlargement of the thymus, which may find confirmation on the Roentgen plate. The shadow of an enlarged thymus is in some respects characteristic. It has the shape of a keystone, with its broad base at the root of the neck, its sides converging slightly as it extends down over the aorta to the heart. Thus in figure 339 is illustrated an enlarged, rather broad thymus in an infant twelve hours after birth, which gave rise to fatal suffocative attacks.



FIGURE 339

Enlarged thymus in infant 12 hours old.

These characteristics are more clearly brought out in figure 340 in which the thymus has the dimensions of a large tumor. In this case also, there were outspoken symptoms of tracheal compression, consisting of cough, crowing respiration and paroxysms of dyspnoea.

Although in the vast majority of cases the normally persisting thymus in infants cannot be seen on the plate we occasionally encounter a large thymic shadow unassociated with any symptoms to suggest disease of this organ. For example, in an infant nineteen days old who suffered a birth fracture of the humerus, a large thymic shadow was accidentally found. (Fig. 341.) The infant was re-examined a number of times during a period of several months, yet no respiratory or other symptoms developed and the thymic shadow remained unchanged. The



FIGURE 340

Large thymus showing typical keystone shape.



FIGURE 341

Large thymus in a 19 day old infant. Accidentally discovered, no symptoms.

absolute clinical latency which may characterize even a very large thymus, is illustrated in figure 341a. In this very young infant there were no symptoms which even remotely suggested the presence of such a large mass in the mediastinum.

Because of the undoubted rarity of thymic enlargement it is proper to interpret critically any shadows in the superior mediastinum of children and to assure one's self that they are not cast by diseased structures other than the thymus. In the case of very young infants the thymic origin of these shadows may be assumed with some assurance not only because of their typical shape but also because of the absence at this age, of other pathological conditions which could give rise to the clinical symptoms.

In children who are more than a few months old however, the interpretation of abnormal masses in the superior mediastinum is rendered less certain because of the increasing frequency of disease of the lymph



FIGURE 341a

Very large thymus in young infant, without symptoms.

nodes which is a more common cause of stridor than enlargement of the thymus. In practice, tuberculosis of the tracheal and bronchial nodes is found so much oftener than is thymic enlargement that the former will have to be excluded before an assumption of thymic disease can safely be made. Tuberculous lymph nodes also produce a wide mediastinal shadow. In most cases this differs in several respects from that of a thymus. The shadow is commonly denser, its borders are convex and owing to the usual involvement of the lower group of nodes, it is widest near the root of the lungs, and narrowest above. (Figs. 112 and 114.) Only in case of enlargement of the paratracheal lymph nodes can there be any confusion with the thymic shadow. These nodes are situated in the superior mediastinum along the lower part of the trachea, in the same situation as the upper pole of the thymus. (Fig. 105.) There is added difficulty in the diagnosis because tuberculosis shows a predilection for these nodes in early infancy and a stridor due to their pressure on the trachea may also be present. The shadow of the paratracheal nodes is however usually unilateral, being especially marked

on the right side and frequently its sharp delimitation below and toward the trachea will serve to distinguish it from the thymus. In doubtful cases, recourse must be had to careful tuberculin tests, which at this early age have considerable value.

In older children, the Roentgen examination may be of considerable importance to the surgeon in the determination of a persistent thymus in cases of so-called status thymo-lymphaticus. There is no doubt that the pre-operative examination of the chest would thus prevent at least some of the sudden and unaccountable deaths in individuals of this bodily habitus. Unfortunately, in most cases of this type, a thymus is either not found by the Roentgen examination or the degree of enlargement is so slight as not to be demonstrable. Equally disappointing is the Roentgen examination for a large thymus in cases of exophthalmic goitre. Although certain investigators have affirmed the frequent enlargement of the thymus in this disease, we have rarely encountered it on the Roentgen plate.

Tumors of the thymus, so called thymomata, may cast a dense shadow in the superior mediastinum which does not differ from other mediastinal new-growths. We illustrate herewith an example of this very rare condition, (fig. 342) with the following clinical history:

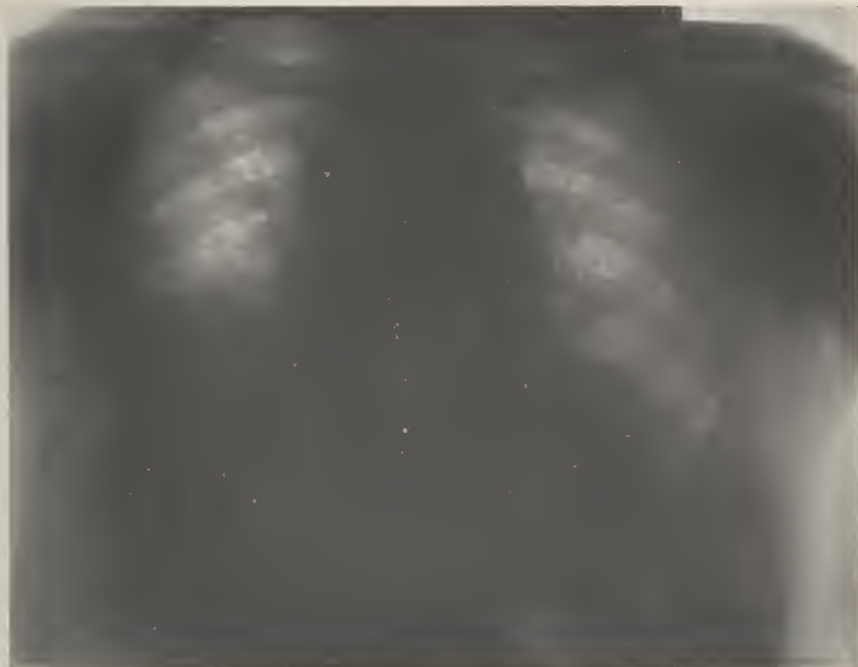


FIGURE 342

Tumor of the thymus. Pleural effusion, right base.

The patient was a boy, sixteen years of age. He became suddenly ill with dyspnoea and oedema of the left arm. The rapid growth of the tumor caused extreme engorgement of the head, neck and chest. Multiple, subcutaneous tumors rapidly developed. Death from suffocation occurred within two months. At autopsy a large tumor mass was found in the superior mediastinum extending from the root of the neck down over the aorta and pericardium. Pathologically, this tumor was a thymoma.

DERMOID CYST OF THE MEDIASTINUM

If we may judge from the small number of cases reported, dermoid cyst is probably the rarest disease of the mediastinum. As is the case with other growths situated deeply in the chest, a dermoid cyst may produce no abnormal physical signs, even when it has grown to a large size, as it may be completely covered by lung tissue. The slowness of its growth also permits the lungs and bronchi to adapt themselves to even a large cyst so that few or no symptoms result. It is only when trauma or infection initiate a sudden growth of the cyst that symptoms arise, the most characteristic of which result from its rupture into a bronchus with the expectoration of its sebaceous or hairy contents.



FIGURE 343

Dermoid cyst of mediastinum. Heart displaced to right. Dorsal view.

The definite Roentgen appearance of a dermoid cyst is in marked contrast to the vagueness of the clinical symptoms. As is illustrated in figure 343 the shadow extends outward from the mediastinum and has a hemispherical shape. Its contour is smooth and well defined. Owing to the nature of the contents of the cyst, the shadow is very dense.

This patient was a young woman whose only symptom for a few years was occasional cough with muroid expectoration. Otherwise she was in good health. Latterly she felt a slight oppression in her chest and there was some increase in the intensity of her cough. The physical examination revealed only a slight increase upward of the area of cardiac dulness. At operation a dermoid cyst the size of a grape fruit was found. Springing from the root of the left lung and growing outward, it occupied the depths of the interlobar fissure and was completely covered by the lung anteriorly. The heart was displaced to the right side.



FIGURE 344

Mediastinal cyst on the right side, extending beyond the mid-line at arrow.

In figure 344 we have a very large mediastinal cyst in a child, which only recently showed signs of rapid growth. In this case the cyst, whose main mass is situated on the right side, also extends beyond

the mid-line so that it is visible beyond the transverse aorta on the left side.

Although, as may be seen, a dermoid cyst casts a definite and in some respects a typical shadow, there are other diseases which are more often responsible for shadows of this kind. We must keep in mind certain massive pulmonary tumors and also encapsulated pleural effusions which occasionally produce a similar shadow. (Fig. 274.) In view of the extreme rarity of dermoid cysts it is therefore wise to have the support of fairly conclusive clinical evidence before the diagnosis is made from the Roentgen examination. Two points of distinction are of especial importance: (1) dermoid cysts are always found in the anterior portion of the chest, and (2) they are encountered most often in early life, especially at puberty when they are prone to take on a rapid growth.

CHAPTER XXIV

Aneurysm of the Aorta

The symptoms and Roentgen shadows of aneurysm are so similar to those of tumors of the lungs and mediastinum that their differentiation from the latter is an ever recurring problem in diagnosis. For this reason aneurysms may profitably be considered in a treatise on the respiratory organs, even though the aorta is anatomically not a part of them.

It is conservatively estimated that fifty per cent of aortic aneurysms are clinically latent or devoid of characteristic physical signs. Physical examination encounters insuperable obstacles in this disease, especially when the aneurysm has not extended to the chest wall; moreover, aneurysms of the descending aorta are so inaccessible to the examiner that they may escape discovery even when they are of large size.



FIGURE 345
Fusiform aneurysm of ascending aorta.

In the majority of cases, the Roentgen appearance of an aneurysm is so typical as to be immediately recognized. Its shadow is superimposed on that of the aorta whose contour it modifies in a number of ways, depending on the size, shape and location of the sacculations or dilatations with which it is affected.

In general we may distinguish two types of aneurysms, the fusiform and the sacculated. These types may exist alone or they may be associated in the same case, as when a sacculum forms in a fusiform aneurysm. The simplest form of aneurysm consists in a fusiform enlargement of a localized portion of the aorta. When this occurs in the ascending arch it is apt to produce few or no objective signs and it attracts attention to itself only because of associated changes in the aortic valves or the coronary arteries. (Fig. 345.) This early stage of the disease is however so commonly latent that when they come under observation, fusiform aneurysms have usually attacked the whole aortic arch. There is then presented the picture of a diffuse dilatation of the entire vessel as in figure 346. It will be noted in this case that

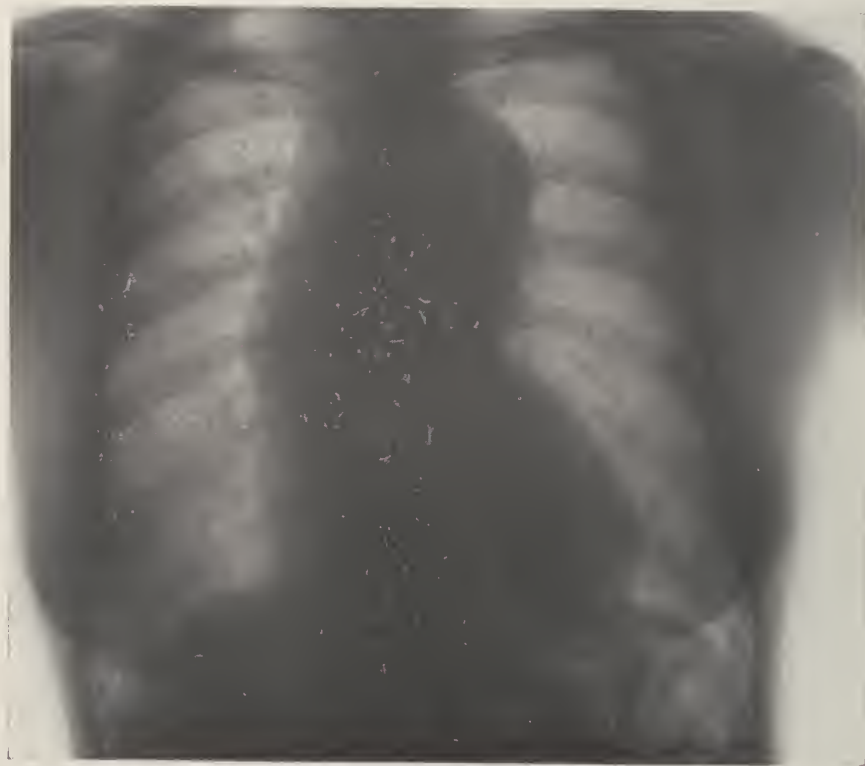


FIGURE 346

Fusiform aneurysm with calcification in the transverse arch.

the contour of the widened aorta is maintained and that the shadow is of great intensity. This is due both to a thickening of the vessel wall and to deposits of lime which are seen in its periphery.

Fusiform aneurysms rarely reach the great size of the sacculated variety, yet figure 347 illustrates the enormous dimensions they may attain. Here also we observe in the wall of the vessel concentric lime deposits and also the slight irregularity of its surface which we may attribute to periarteritis. These aneurysms bear intrinsic evidence of their age and the density of the shadows which they cast testifies to the rigidity of their walls.

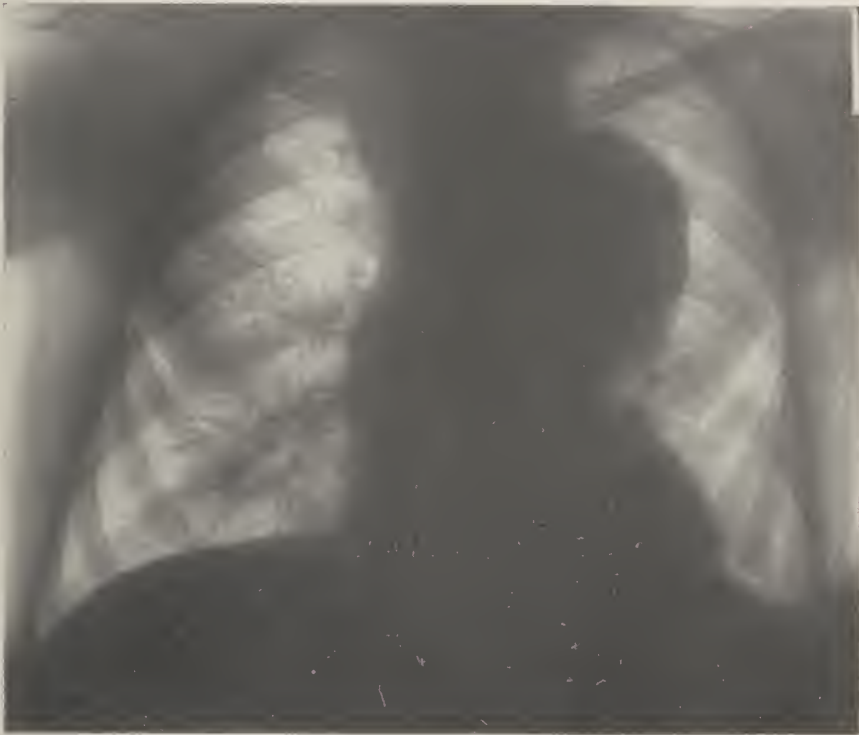


FIGURE 347

Large fusiform aneurysm with concentric lime deposits. Aneurysm of innominate artery. Periarteritis.

When aneurysms have grown to a considerable size they rarely preserve the uniform contour noted in the cases so far described. They tend, in some portion of the aorta to increase in size disproportionately to the others so that localized prominences or sacculations are produced. The resulting shadow masses may assume a great variety of shapes.

Because of a predilection of the syphilitic process for the root of the aorta and also because this part of the vessel receives the first impact of the blood, the sacculations are usually most prominent in the ascending arch. As they grow, they extend upward, outward and frequently forward so that they encroach on the right lung.

A simple form of sacculation of the ascending arch is illustrated in figure 348, in which apparently the rest of the aorta has remained free of dilatation. The mass of the aneurysm in this case grew backward and laterally so that it did not come into contact with the thoracic wall. For this reason it produced no physical signs. As is so often the case with aneurysms it manifested itself by symptoms referable to other organs within the chest, in this case, the lungs. Repeated small hemoptyses which aroused a suspicion of tuberculosis led to the Roentgen examination and the unexpected finding of the aneurysm. The slow growth of this aneurysm, unassociated with any further symptoms is brought out in figure 349 which represents the condition three years later.

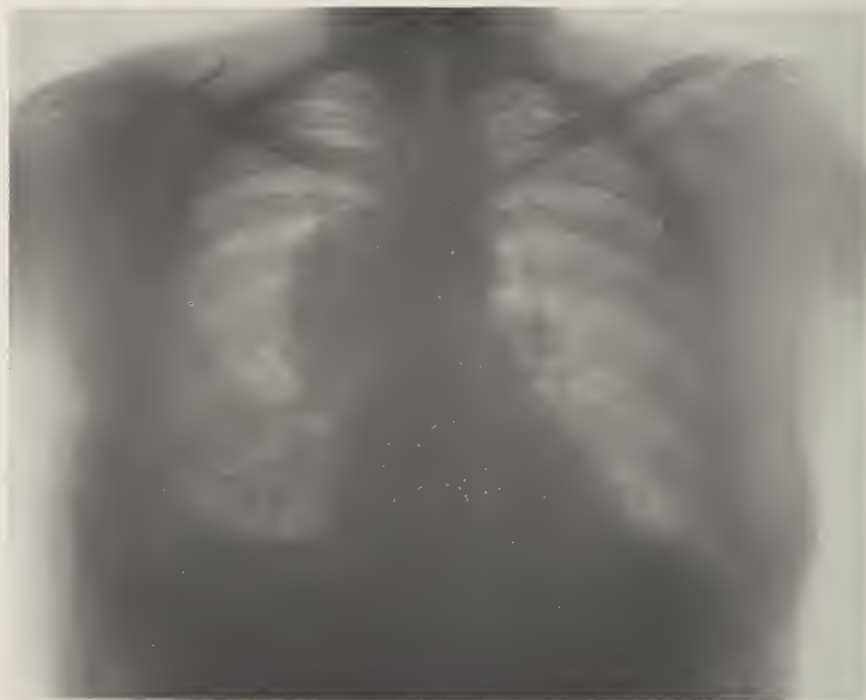


FIGURE 348

Sacculated aneurysm of ascending arch. Only symptom, recurring hemoptysis. See figure 349.



FIGURE 349

Same as figure 318, three years later, showing slow growth of aneurysm.

Usually aneurysms of the first part of the aorta grow upward; they may however in exceptional cases extend downward and over-hang the right auricle. (Fig. 350.) In this case the proximity of the aneurysm to the aortic valves and their involvement in the syphilitic process resulted in a valvular insufficiency.

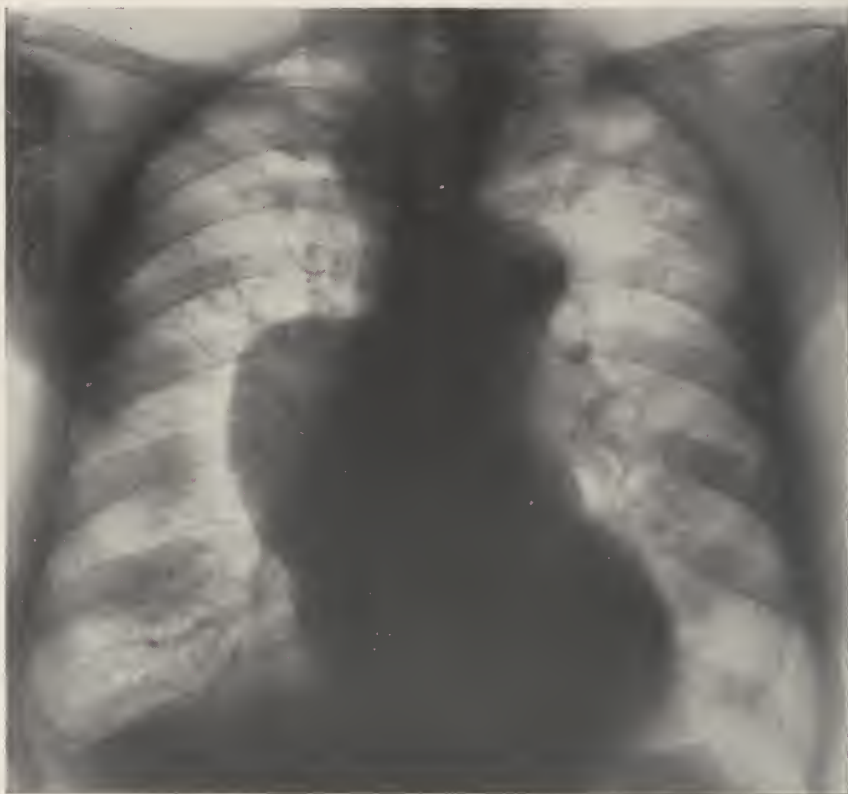


FIGURE 350

Sacculated aneurysm of ascending arch, extending down over right auricle. Aortic insufficiency.

Sacculations of the aorta are more often multiple than single. Thus in figure 351 there are dilatations both of the ascending and transverse arch. In this case also the greater size of the aneurysm of the ascending arch speaks for the earlier involvement of this portion of the vessel. The great diversity in the size, shape and situation of the sacculations is brought out in figures 352, 353 and 354.

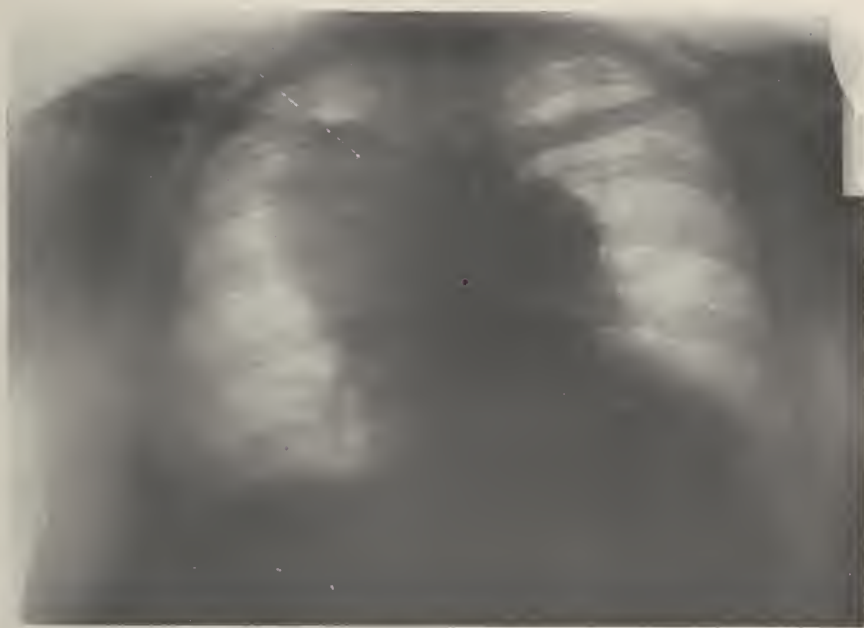


FIGURE 351

Sacculated aneurysm of the ascending and transverse arch of the aorta. Physical signs of mediastinal tumor.

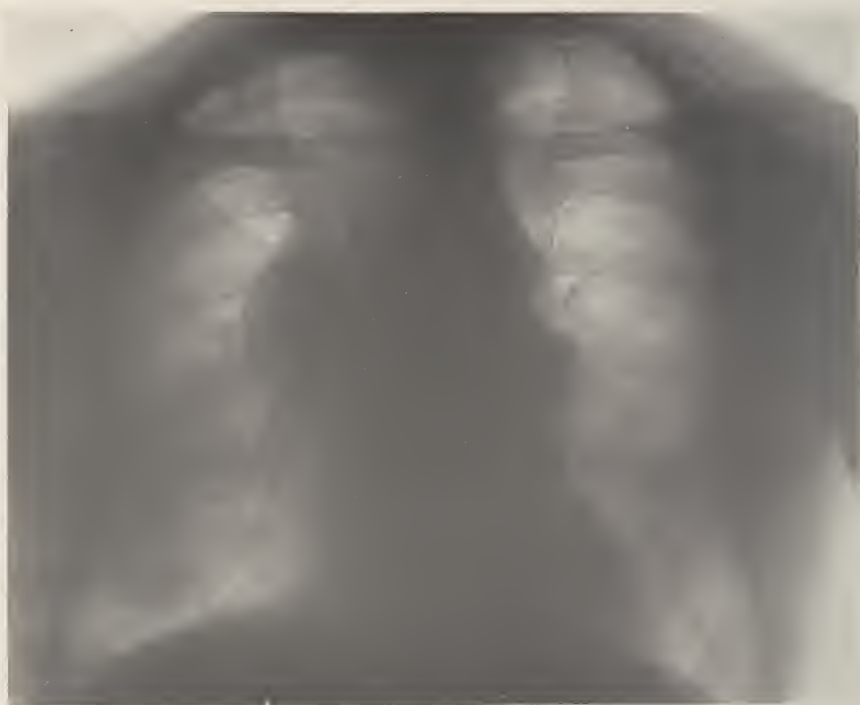


FIGURE 352

Sacculated aneurysm of ascending and descending arch of aorta. Trachea displaced to right.



FIGURE 353

Sacculated bilocular aneurysm of ascending and descending arch.



FIGURE 354

Sacculated aneurysm with depression of transverse arch of aorta.



FIGURE 355
Aneurysm of transverse arch.

Solitary aneurysms of the transverse arch (fig. 355) are less common than those thus far described; they may attain an enormous size. (Fig. 356.) Aneurysms of the descending aorta are perhaps the most difficult of diagnosis by the ordinary clinical means. They are inaccessible to examination and for this reason, they may remain latent even though of considerable size. In figure 357 is illustrated a large fusiform aneurysm of the descending aorta which gave no direct symptoms or physical signs. The dense shadow, extending to the left of the heart, showed a marked systolic pulsation; viewed in the oblique position, its continuity with the descending aorta was demonstrated. An aneurysm of this type may be the cause of symptoms which are mistakenly referred to the oesophagus or stomach and it is therefore apt to be discovered by the gastro-enterologist, as was the case with this patient who complained of vague gastric symptoms.



FIGURE 356

Sacculated aneurysm of transverse arch.



FIGURE 357

Fusiform aneurysm of descending aorta. Clinically latent.

Aneurysms of the lower part of the thoracic aorta present extreme difficulty in diagnosis. They may be completely obscured by the heart and require a fluoroscopic examination for their certain recognition. They are best seen in the oblique positions.

In addition to intrinsic changes in the wall of the aneurysm such as peri-arteritis and calcification, the Roentgen plate occasionally discloses a weakening of the arterial wall at one point which may be suggestive of an impending perforation. Such a perforating aneurysm is shown in figure 358. The small semicircular prominence, superimposed on the body of the aneurysm, indicated, by the thinness of its wall and its excessive pulsation, the imminence of rupture at this point.

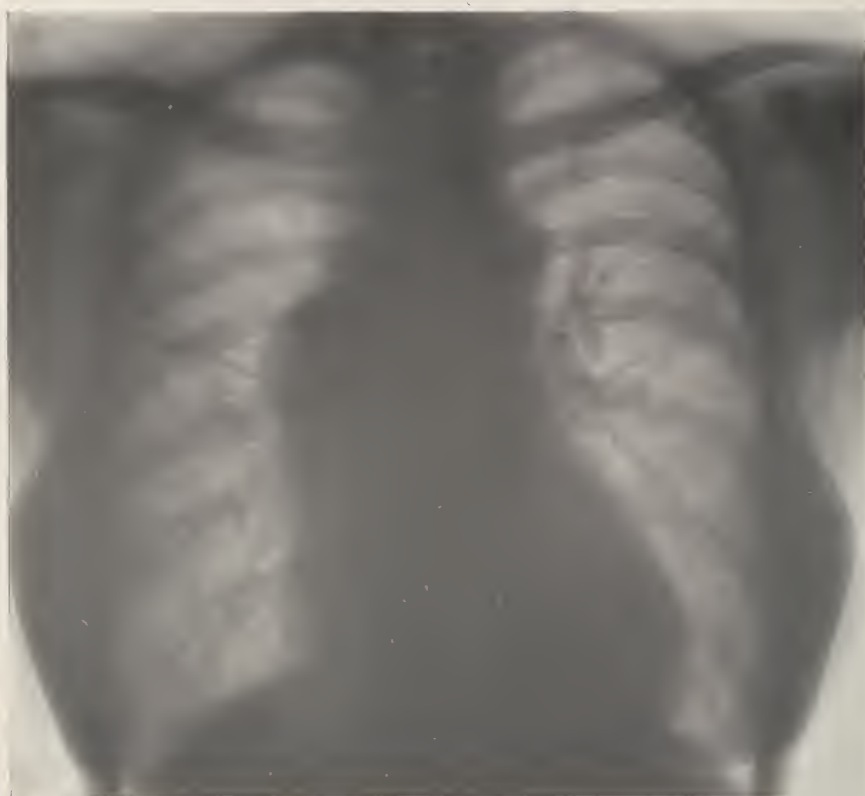


FIGURE 358

Fusiform dilatation of aorta. Secondary dilatation of ascending arch.

PRESSURE EFFECTS OF ANEURYSMS

Aside from the actual demonstration of the aneurysm, the Roentgen examination supplies instructive facts in regard to its pressure effects on the other thoracic organs. For this purpose, fluoroscopic examination is of especial value, as it enables us to observe the relations of the aneurysm when viewed from various directions. The origin of many of the symptoms which arise in the trachea, bronchi and oesophagus thus become manifest to us and occasionally we may observe an encroachment on, or even a perforation of the bony thorax.

Perhaps the most frequent pressure of effect of aneurysms especially when they are sacculated, is exerted on the trachea, which may suffer an extreme constriction. The trachea may be flattened ventro-

dorsally against the spine; usually however, it is compressed laterally between adjacent sacculations. Its uneven lumen may then be seen as a light area traversing the dense shadow of the aneurysm. The details of malposition and distortion of the trachea may at times be best seen in the oblique positions. Thus, in figure 359 the large globular aneurysm had effected a marked narrowing and displacement of the trachea whose lumen is almost completely obliterated.



FIGURE 359

Uniform dilatation of entire aortic arch.

Pressure on one of the larger bronchi, which is a frequent result of aneurysm is not directly visible but may often be inferred from a diminished air content of the affected portion of the lung. If the aeration of a sufficiently large portion of the lung is affected, a broncho-stenotic phenomenon may be visible on the screen; during inspiration there occurs a displacement of the mediastinum toward the affected side. Gross pulmonary changes, resulting from a long-standing narrowing of a bronchus, such as bronchiectasis and abscess of the lung which, because of their similarity to tuberculosis have become known clinically as aneurysmal phthisis are, of course, readily discernible on the plate.

Compression of the oesophagus occurs less often than that of the trachea; it may however be marked and be the first symptom to draw attention to the aneurysm. The patient whose aneurysm is illustrated in figure 360 complained mainly of dysphagia, which caused him much distress. The degree of narrowing may be judged from the thin column of barium in the oesophagus; the tortuous course of the latter over the surface of the aneurysm is also evident.



FIGURE 360

Oblique view of aneurysm of transverse arch of aorta showing constriction of trachea. Marked tortuosity of oesophagus indicated by ingested barium at arrows.

DIFFERENTIAL DIAGNOSIS

Although most aneurysms may be recognized as such immediately by means of a Roentgen examination, occasionally the shadows are so similar to those of new growths of the mediastinum, that all our resources, both clinical and Roentgenological are taxed to make the distinction. Of course, in doubtful cases the value of a Wasserman reaction cannot be over-estimated and a positive test should outweigh the Roentgen findings even when the latter are suggestive of a new-growth. Of the greatest service in such doubtful cases is the thorough fluoroscopic examination of the patient, in every possible position. This

procedure will usually determine the relation of the shadow to the aorta. The great similarity which some mediastinal tumors may bear to aneurysms will be evident by a study of the new growths illustrated in figures 324 and 325.

The following points of distinction between aneurysms and new growths may be pointed out:

1. The outline of an aneurysm is smooth and curved, whereas tumors are frequently lobulated and their borders may be irregular. The latter may also invade the lung so that their outlines become indistinct.

2. The shadow of a tumor can frequently be distinguished from that of the aorta which it overlies, as it is usually of lesser density. It may even displace the arch of the aorta downward by pressure on it.

3. Examination of the patient in the oblique position may show that a questionable shadow is not a part of the aorta. For this purpose, fluoroscopy in the second oblique position is of special value, as it visualizes the aorta throughout a great part of its course. In this position only, can the smaller aneurysms which occasionally form in the concavity of the aortic arch, be brought to light.

4. Evidence of aortic valvular insufficiency with dilatation of the left ventricle will favor aneurysm.

In spite of these points of distinction, there are occasional cases of tumor of the mediastinum which cast shadows that are absolutely like those of undoubted cases of aneurysm. In these patients, the physical signs, unfortunately, are of little help as they are apt to be identical in both conditions and we are forced to observe the further progress of the case before we may reach a decision. Aneurysms are of slow growth, whereas tumors increase in size more rapidly; a second Roentgen examination after a short interval may therefore settle the question. The discovery of a pleural effusion during the progress of the case or the sudden swelling of an arm due to thrombosis of the subclavian vessels will incline the examiner to a diagnosis of new growth because of the tendency of tumors to produce vascular compression with subsequent thrombosis. Aneurysms, on the other hand, even when they are very large, are rarely responsible for these symptoms.

PULSATION OF ANEURYSMS

It is customary, in making a differential diagnosis between aneurysm and tumor, to emphasize the importance of a pulsation of the shadow, as an evidence of the former. It is true that a systolic pulsation may be regarded as a valuable corroborative sign of aneurysm. But it is not present in every case or even in a large percentage of the

cases and for this reason it is not a dependable sign. Even very large aneurysms may not show the faintest evidence of pulsation. The causes for this may be various. In the first place, the wall of the aneurysm is often thickened by disease and by the deposit of a lamellated clot, whose rigidity interferes with the transmission of pulsations. Further, in sacculated aneurysms, the communication with the main channel of the aorta may be a small one and the impulse of the stream of blood may not be strongly felt in the aneurysm. In the fusiform variety, for a contrary reason, the pulsation is usually well marked.

In nearly all cases however, the pulsation, if it is at all visible, is of no greater amplitude than in the normal aorta. The pulsation seems to be greater on the screen because of the larger size and density of the aneurysmal shadow, which make it more obvious. The reasons for this absence of marked pulsation are readily understandable. As will be pointed out, in most cases of aneurysm there is no enlargement of the heart and therefore during systole, the aneurysm is not distended with a greater amount of blood than is the normal aorta. It is only when vascular disease affects the root of the aorta and implicates its valves, that the resulting aortic insufficiency with its increased stroke volume produces a wide expansile pulsation of the vessel. Under these circum-

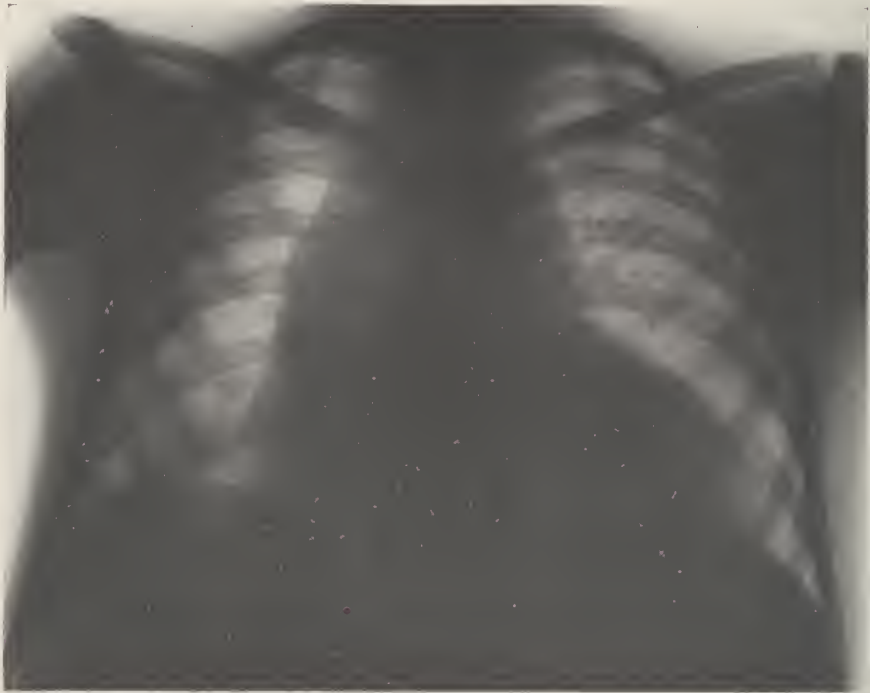


FIGURE 361

Syphilitic aortitis with diffuse dynamic dilatation of aorta. Aortic insufficiency and congenital syphilis.

stances, the aorta may exhibit a marked pulsation without being much dilated. This is strikingly illustrated in figure 361. The enlarged shadow of the aorta is entirely the result of a dynamic dilatation produced by the vigorous contraction of the hypertrophied and dilated left ventricle. At autopsy there was found congenital syphilis of the ascending arch of the aorta with an aortic insufficiency. The aorta itself was not dilated.

Finally it must be borne in mind that tumors of the mediastinum occasionally pulsate. This may be due to a vascularity of the tumor itself; more often it is a transmitted pulsation from the aorta. Although such a transmitted pulsation has not the expansile character of an aneurysm, the determination of this point on the fluoroscopic screen is not always an easy matter.

THE HEART WITH AORTIC ANEURYSMS

Aneurysm of the aorta by itself has no influence on the size of the heart. When the latter is found to be enlarged, this must be attributed to other associated conditions rather than to the aneurysm. The commonest of these is aortic insufficiency due to an involvement of the valves in the syphilitic process or chronic nephritis with arterial hypertension. Occasionally a large aneurysm of the ascending aorta will displace the heart to the left by downward pressure; this must of course not be confused with a cardiac dilatation.

Despite these facts in regard to the size of the heart which we have verified by numerous orthodiagraphic measurements, there is a more or less prevalent belief that aneurysms cause an enlargement of the heart. This belief, to which the Roentgen examination has in a measure contributed, is due to the failure to distinguish between a true aneurysm of the aorta and the very common atherosclerotic dilatation of this vessel. In the latter condition, in which there is often an arterial hypertension the heart is frequently hypertrophied and dilated and the elongated transverse arch of the aorta extends prominently into the left pulmonary field. Only those lacking experience will interpret such an enlargement of the aorta as an aneurysm. It is an almost universal phenomenon of senescence whose familiar features receive no more than casual notice from the experienced clinician or Roentgenologist. The distinction between the atheromatous and the aneurysmal aorta on the Roentgen plate is as definite as that between the corresponding degenerative and luetic changes in the vessel wall; with the former, the heart is often hypertrophied and dilated, with the latter, it is rarely so.

ANEURYSM OF THE INNOMINATE ARTERY

Aneurysms of the innominate artery are usually associated with a similar disease of the aorta. When they are sufficiently large they cast a shadow which begins at the summit of the aortic arch and extends to the sternal end of the clavicle. Such an aneurysm is illustrated in figure 347, in which the dilatation of the innominate is an extension of a general aortic dilatation. In figure 362 the aneurysm appears to be a sacculated one.



FIGURE 362

Aneurysm of innominate artery.

Rarely, the innominate artery is involved alone and it may then appear as a semicircular shadow situated below the inner end of the clavicle, just above the aorta. This shadow bears a close similarity to that of a unilateral substernal thyroid, (see figure 329,) from which it can be distinguished on the screen by its pulsation and its failure to move during the act of swallowing.

In patients with atheroma of the aorta, it is not unusual to feel pulsating vessels in the suprasternal notch or behind the insertion of the sterno-mastoid muscle. The Roentgen plate in these cases shows usually that the aorta is elongated so that its large branches become more accessible to palpation at the root of the neck. The latter are also atheromatous and cast a wider shadow than is normal in the superior mediastinum. They can, however, readily be distinguished from true aneurysms of the innominate artery.

SECTION VII

The Intrathoracic Lymph Nodes

Infectious Adenopathies

Non-Infectious Adenopathies

Glandular Tumors

Lymphomata

Hodgkin's Disease

CHAPTER XXV

The Intrathoracic Lymph Nodes

INFECTIOUS ADENOPATHIES

The bronchial lymph nodes, by which we mean in general the lymphatic tissue into which the pleural, pulmonary and bronchial lymphatics drain, bear the brunt of numerous and various injuries during the life of the individual. It is not surprising in view of the common and repeated respiratory infections, that the lymph nodes are almost universally enlarged and diseased. However common this bronchial adenitis is, in adults at least it maintains a subordinate place in the clinical picture and rarely by itself is responsible for any recognizable symptoms.

We may dismiss acute bronchial adenitis with a few words. The bronchial nodes are undoubtedly enlarged as a consequence of acute infections of the bronchi and lungs. In pneumonia for example, the hilum shadows are noticeably increased in size and a similar hyperplasia is commonly seen during or after measles and whooping cough. The glandular enlargement, however, is usually moderate and it has no clinical significance. In infants and young children we may, on the other hand, observe a tendency to a more vigorous reaction of the lymphatic apparatus which is similar in its intensity to that seen in infantile glandular tuberculosis. The nodes may attain enormous proportions, so that they resemble a tumor mass; yet with the subsidence of the infection, they return to their usual size with astonishing rapidity. For example, in figure 363 is illustrated an acute adenitis of the upper and lower groups of bronchial nodes which developed during the course of an unrecognized retropharyngeal abscess in a young child. A week later, after the evacuation of the abscess, the lymph nodes had practically disappeared.

Of greater importance are the adenopathies which result from the various chronic respiratory infections. Rarely in adults do we see a plate without some evidence of glandular disease at the roots of the lungs. It may be stated without exaggeration, that in the anatomic sense, a perfectly normal lung root does not exist, because practically no individual can escape the glandular irritation due to dust inhalation and the various bacterial infections including, above all, tuberculosis.



FIGURE 363

Acute bronchial lymph-adenitis secondary to retropharyngeal abscess. Note shadow of lower group of nodes behind the heart. Nodes disappeared in a few weeks.

There is perhaps no subject which has given rise to more confusion in the interpretation of chest plates than chronic disease of the bronchial lymph nodes. This is mainly owing to the failure of Roentgenologists to distinguish between infection and clinical disease. The dense, irregular shadows which are commonly seen at the roots of adult lungs are undeniably due to an enlargement of the lymph nodes; but it is another matter, in a case of thoracic disease, to regard them as related to the disease. In fact, in adults at least, it is questionable whether such enlarged lymph nodes, which are usually indurated and anthracotic, contribute anything to the clinical picture. The cough and expectoration which may be present in these cases, are more logically referable to an underlying bronchitis, which causes both the symptoms and the glandular enlargement. We may often find these symptoms together with glandular swelling as a result of chronic accessory sinus disease. (Fig. 364.)



FIGURE 364

Hyperplasia and induration of bronchial nodes secondary to chronic nasal sinusitis.

These considerations have a most important bearing on our interpretation of the shadows of tuberculous bronchial lymph nodes. The changes in these nodes by which we recognize them as tuberculous are essentially caseation and calcification, which cause the shadows to become very dense and irregular. (Figure 365.) These changes may be noted in the broncho-pulmonary and tracheo-bronchial nodes and in adults very rarely in the paratracheal nodes. The glandular enlargement is always moderate and never attains the dimensions of the tuberculous adenopathy in children. We must now ask ourselves what significance from a clinical standpoint is to be attached to the discovery on a Roentgen plate of a number of enlarged tuberculous nodes at the roots of the lungs. The clinician has repeatedly presented to him the problem of a patient who has a slight daily rise of temperature, with or without cough and in whom the Roentgen plate reveals only the shadows of presumably tuberculous bronchial nodes. The question



FIGURE 365

Calcification of tuberculous bronchial lymph nodes at right root.

arises whether such nodes are a sufficiently common cause of pulmonary or other symptoms that their presence in an individual case will warrant us in assuming that they are the cause of those symptoms. Decidedly not. Glandular tuberculosis is almost universal in adults, yet it is practically always a quiescent infection which gives rise to none of the clinical symptoms of tuberculosis. If, in a given case, no infiltrations are seen in the lungs, the examiner will wisely disregard the enlarged lymph nodes and will look elsewhere than in the chest for the cause of the fever. The Roentgen examination in this type of case will there-

fore be of value in a negative sense, in that it will serve to exclude tuberculous disease of the lungs and direct attention to other foci such as the teeth or the tonsils or the accessory sinuses.

In regard to syphilis of the intrathoracic lymph nodes little that is definite can be said. Whether the Roentgen shadows of enlarged nodes should in a particular case be regarded as syphilitic will depend less on the appearance of the shadows themselves than on the evidences of syphilis elsewhere in the body which will lend plausibility to the diagnosis. In the two cases herewith illustrated the shadows have the faint homogeneous appearance which we found in the various lymphomata. (Figs. 366 and 367.)



FIGURE 366

Enlargement of right paratracheal lymph node in syphilis.



FIGURE 367

Enlarged bronchial lymph nodes in a syphilitic.

NON-INFECTIOUS ADENOPATHIES

GLANDULAR TUMORS. The primary tumors of the intrathoracic nodes belong mostly to the group of lympho-sarcomata. In our discussion of mediastinal tumors we have already illustrated the Roentgen features of these cases. The disease may be exclusively limited to the intrathoracic nodes or the latter may be only a local manifestation of a general lympho-sarcomatosis. In either case, the lymphatic enlargement may be extreme and have the dimensions and produce the pressure effects of a tumor. There are cases of general lympho-sarcomatosis on the other hand, in which the bronchial nodes play a subordinate role; they show a moderate enlargement and cast the homogeneous, well defined shadow so characteristic of new-growths,

yet they never attain the size of tumors and produce no symptoms. It is none the less important to recognize the significance of these smaller shadows as the existence of intrathoracic disease has to be reckoned with in the treatment of these cases.

Tumors of the bronchial nodes which are secondary to primary growths in the lungs are of little clinical importance. They are usually not very large and they are entirely over-shadowed on the Roentgen plate by the primary tumor. There are, however, occasional exceptions to this rule. A small primary carcinoma of a bronchus may itself be of slow growth. Its metastases in the adjacent nodes may, on the contrary, grow so rapidly that they quickly form a large mediastinal tumor, which may be responsible for most of the clinical symptoms. (Fig. 319.)

Metastatic involvement of the bronchial nodes is a not uncommon sequel of carcinoma of the breast. This is accounted for by the direct communication between the subpectoral and intrathoracic lymphatics. In spite of the frequency of this complication, the Roentgen plate does not often throw light upon it. Cancer cells transplanted to these nodes are usually of such slow growth that a significant enlargement of them is not often seen; for this reason a negative Roentgen plate is no assurance that the lymph nodes are not involved. Only when, as we have observed in an isolated case, an enlargement occurs in an unusual situation, as in a paratracheal node, may we attach any significance to it. Often as not, the first indication of an intrathoracic complication will be a pleural effusion which may occur months or years later, or as we have seen, it may take the form of a wide spread pleuro-pulmonary carcinosis.

LYMPHOMATA. In the various blood diseases the intrathoracic lymph nodes are frequently involved together with the glandular apparatus elsewhere in the body. The enlargement of the bronchial nodes may take one or two forms. It may be extreme, the individual nodes growing to an enormous size so that on the Roentgen plate they produce the appearance of a true mediastinal tumor. Thus in figure 322 the lymphoma assumes the form of a smooth, dense tumor mass, whose pressure has resulted in a pleural effusion. In figure 326 the broncho-pulmonary nodes on the left side are especially enlarged; they occupy the position of the pulmonary artery, with which they could readily be confused. In both of these cases, the clinical picture was that of a chronic lymphatic leukemia. In acute leukemias, a similar though more rapid enlargement of the bronchial nodes may occur. Thus in a case of acute myeloblastic leukemia, the nodes completely filled the superior mediastinum and also encroached on an upper lobe producing marked respiratory embarrassment. (Fig. 368.)



FIGURE 368

Enlargement of upper mediastinal lymph nodes in acute myeloblastic leukemia.

These however, are exceptional cases; in most cases of leukemia the bronchial adenopathy remains within moderate limits. The root shadows are somewhat enlarged and present the faint, homogeneous, well outlined appearance which is so typical of lymphomata in general, especially Hodgkin's disease. (Fig. 369.) In many instances the enlargement is so slight that one cannot readily distinguish it from the hyperplastic anthracotic nodes which are such a regular feature of the Roentgen plate in adults. Leukemic infiltrations in the lungs are rarely found. When present, they closely resemble areas of lobular pneumonia, whose confluence may produce an appearance of consolidation of the lung. (Fig. 370.)



FIGURE 369

Enlargement of bronchial lymph nodes in acute leukemia.

In conclusion, it may be worth while to consider in general the clinical application of the Roentgen examination of the thoracic lymph nodes. The clinician is repeatedly brought face to face with cases whose essential feature is an enlargement of one or more groups of superficial lymph nodes. In many instances, the character of the blood changes or other associated abnormalities at once affords the key to the nature of the glandular enlargement. There are, however, other cases in which such aids in diagnosis are wanting. The examiner will then have to be guided by the physical characteristics of the enlarged lymph nodes themselves. Unfortunately these are not always determinant. It is not easy at times to distinguish between the glandular enlargement of Hodgkin's disease, lympho-sarcoma or benign lymphomata and that due to chronic infections such as tuberculosis and syphilis.



FIGURE 370

Chronic lymphatic leukemia. Enlargement of bronchial nodes and leukemic infiltration of left lower lobe.

In this dilemma the Roentgen examination of the chest may occasionally shed decisive light on the true condition. The Roentgen appearance of intrathoracic Hodgkin's disease is often typical. In other cases, the discovery of a large mediastinal mass will speak for the sarcomatous nature of the superficial lymphomata. And finally, a tuberculous lesion in the lungs, especially if it is recent, may incline one, though not necessarily so, to suspect that the lymph nodes felt on the surface of the body are tuberculous.

CHAPTER XXVI

Hodgkin's Disease

Although the symptoms and mechanical effects of intrathoracic Hodgkin's disease are essentially those of true mediastinal tumors, it may nevertheless claim special consideration from the Roentgen standpoint. The Roentgen manifestations of this disease are manifold and they present some unique features which are of value in distinguishing it from malignant tumors and other diseases within the chest.

Of course, Roentgen examination of the chest is rarely necessary in order to establish the diagnosis of Hodgkin's disease, because external lymphomata are usually present. Yet it cannot be a matter of indifference in the individual case whether the intrathoracic lymph nodes are also involved or the lung is actually invaded by the disease. From the point of view of treatment also, it is evident that therapeutic measures applied exclusively to the external glandular enlargement can secure no permanent results, if the disease has invaded the chest.

The bronchial nodes are a frequent site for the development of this disease. In nearly all cases in which the glandular enlargement in the neck or axilla was well developed, we have found on the plate definite changes either at the roots of the lungs or in the lungs themselves. More than this, in those uncommon forms of the disease in which only the internal organs are affected, we have almost invariably found pathological changes in the thoracic viscera, from which at times the nature of the process in the abdomen might be inferred.

The value of the examination of the chest therefore requires no further comment. One other consideration is worthy of note. Only the Roentgen examination is capable of disclosing the pathological changes which are so frequently present in the chest in cases of Hodgkin's disease. The physical signs are rarely decisive even when large tumor masses are present and the clinician is usually dependent on the symptoms which are often absent and on the signs of pressure on the bronchi or vessels for evidences of the disease.

The abnormal shadows found on the plates in cases of Hodgkin's disease permit of a classification into four groups, the features of which may be found singly in the individual case or as is more common, they may be combined.

(1) Among the rarer Roentgen types are those that resemble true mediastinal tumors, especially the lympho-sarcomata. The plate reveals a dense central shadow, the outlines of which may be irregular and lobu-

lated and in which all evidences of individual lymph nodes are obliterated. (Figs. 371 and 372.) The patients suffer the symptoms of intrathoracic pressure which are associated with tumors and the plate offers no point of distinction from them. This form of the disease may be combined with the other types.



FIGURE 371

Hodgkin's disease. Mediastinal tumor type.



FIGURE 372

Mediastinal tumor. Hodgkin's disease.

(2) The central lymphomatous mass may extend outward into the lung and infiltrate it extensively. The contour of the growth loses its sharp definition, penetrating into the lung parenchyma and shading off into it imperceptibly. (Fig. 373.) In this infiltrative form, the

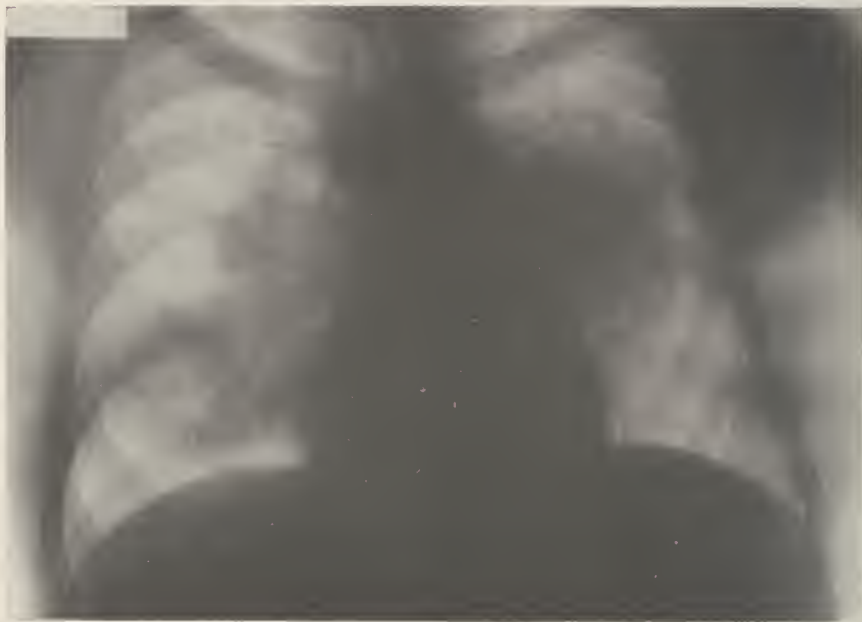


FIGURE 373
Hodgkin's disease. Unusual infiltrating form.

tumor masses may be continuous with the central lymphoma or there may be in addition isolated deposits scattered throughout the lung, the outlines of which are also indistinct. (Fig. 374.) More rarely the infiltration may be a diffuse one and involve a whole lobe in an irregular consolidation, somewhat like a pneumonia. (Fig. 377.)



FIGURE 374

Hodgkin's disease. Diffuse infiltration of both lungs.

As in the previous form the shadows here have the denseness of a true neoplasm and it may perhaps be inferred that we are dealing with longer standing cases in which there has been a formation of considerable connective tissue. This also is one of the less common forms of the disease in which there is little that is typical to lead the Roentgenologist to a correct diagnosis.

(3) Among the more characteristic features of the disease we must regard the circumscribed isolated deposits of granulomatous tissue which may be found anywhere in the lung. They occur as smaller or larger irregularly circular shadows of very slight density, so slight in fact that the lung markings are distinctly visible through them. In some respects these shadows resemble those of the metastases of malignant growths; they differ from them however, in that they lack the perfectly circular outline of cancers and are often grouped in lobulated masses.

In figure 375 these isolated deposits are situated mostly near the roots of the lungs; yet others are found near the periphery and they may even occur near the apex. Here, if viewed alone and without reference to other evidences of the disease, they may readily be confused with the early infiltrations of pulmonary tuberculosis. (Fig. 376.)



FIGURE 375

Hodgkin's disease. Enlargement of bronchial nodes. Isolated deposits in lungs. Involvement of substernal thyroid.



FIGURE 376

Hodgkin's disease. Characteristic involvement of bronchial and paratracheal nodes. Isolated deposits in both lungs.

This form of Hodgkin's disease does not appear to occur by itself but is associated with its other forms. The isolated position of the deposits in the lung would indicate, in accordance with the accepted view of the pathology of the disease, their autochthonous development from the foci of lymphoid tissue which are found throughout the lungs.



FIGURE 377

Hodgkin's disease. Very large paratracheal nodes. Lymphomata at left root. Diffuse infiltration of right lower lobe.

(4) The common and typical change in the chest in Hodgkin's disease consists in a tumefaction of the intrathoracic lymph nodes, which is analogous to that found in the neck or in the axilla. In a large percentage of cases in which the disease is well developed there will be found at the roots of the lungs the shadows cast by these lymph nodes which in some respects are characteristic. They are well defined, faint in intensity and homogeneous. There is usually no difficulty in distinguishing them from the lymph nodes of tuberculosis or anthracosis, as the latter produce the well known dense irregular shadows which are so commonly found at the hilum of the lungs. The outline of the individual nodes is often maintained, a circumstance which accounts for the large, lobulated masses such as are seen in figures 375 and 378.

Of especial interest in this type of Hodgkin's disease, is the remarkable predilection shown for the enlargement, at times enormous, of a group of lymph nodes which is rarely affected in other diseases. In over fifty per cent. of the cases which have come under our observation, there has been a marked increase in the size of the paratracheal group



FIGURE 378

Hodgkin's disease. Characteristic enlargement of bronchial and paratracheal nodes.

of nodes, either alone or associated with involvement of other intrathoracic lymphoid tissue. The great size which these may attain is illustrated in figures 376, 377 and 378. They cast a shadow on the plate which is situated to the right of the trachea, extending from its bifurcation up to the sterno-clavicular articulation. Their deep situation makes them inaccessible to the examiner and they produce no superficial physical signs. The frequency with which this paratracheal adenopathy is found in Hodgkin's disease makes the association more than a coincidence and for this reason we regard it of major importance in diagnosis. It occurs with the greatest rarity in mediastinal and pulmonary new-growths and then the enlargement is usually very slight. We have found a marked enlargement of these nodes occasionally in other diseases which affect the intrathoracic lymphoid tissue such as lympho-sarcoma and lymphatic leukemia and also the glandular tuberculosis of infants and young children. In fact, tuberculosis of the bronchial lymph nodes in children may present a Roentgen appearance which is identical with that of Hodgkin's disease.

The adenopathy of Hodgkin's disease can readily be distinguished from that of tuberculosis in adults. When tuberculous lymph nodes have attained the size of those in Hodgkin's disease, they invariably show evidences of caseation or calcification and they are so matted together that the outlines of individual nodes is obliterated. For this reason, large lobulated shadows at the roots of the lungs, which are faint and homogeneous, will raise a strong presumption of Hodgkin's disease.

Although it will appear from the foregoing that Hodgkin's disease exhibits itself in protean forms, some of them are distinctive and may be utilized for diagnosis when the rest of the clinical picture is incomplete or atypical. There come to mind the cases of abdominal Hodgkin's disease in which the clinical picture may be confused by the high remittent fever; or again the so-called splenomegalic form in which the external glandular enlargement may be insignificant. In these cases especially, the Roentgen examination may bring to light a large mediastinal mass the existence of which was unsuspected. This is well illustrated in figure 378. This patient presented the clinical appearance of a severe secondary anemia and complained chiefly of dragging pain in the left side due to the weight of an enormous spleen which extended down to the pelvis. The only palpable glandular enlargement was to be found in the left supraclavicular fossa where one small hard lymph node could be felt. Yet the plate shows that the disease had already extensively involved the intrathoracic nodes, notably the right paratracheal group.

The clinical distinction between glandular tuberculosis and Hodgkin's disease may at times be so difficult that only a microscopic examination of the diseased nodes will clear up the case. Here also the Roentgen examination may play a role of decisive importance in revealing suggestive, if not characteristic, changes within the chest.

In interpreting the shadows seen on the plates of patients whose external nodes show the pathological changes typical of Hodgkin's disease, it must not be forgotten that occasionally the disease within the chest may be of an entirely different nature. Glandular tuberculosis has at times been found in close association with Hodgkin's disease, involving either the same or different groups of nodes. Of the unusual combinations we may instance a terminal miliary tuberculosis of the lungs the shadows of which were later seen in the case illustrated in figure 378 and a mediastinal lymphosarcoma (fig. 315) in a patient whose external lymphomata were of the Hodgkin's type.

SECTION^e VIII

The Diaphragm

CHAPTER XXVII

The Diaphragm

Our interest in the diaphragm from the Roentgen standpoint, arises less from disease of this muscle itself than from its unique position. Interposed between the thoracic and abdominal viscera, it may reflect, by an alteration in its position and contour, abnormal conditions both in the chest and in the abdomen. It therefore becomes a matter of importance in cases of visceral disease to scrutinize the diaphragm in respect to its position, shape and movement.

Under normal conditions the diaphragm exhibits a great diversity in shape and position and in the range of its movement. These variations seem to be most influenced by the shape of the chest. In long-chested individuals, whether they are so congenitally or as a result of emphysema, the diaphragm is low and its contour flattened so that the costo-phrenic sinus is shallow. This condition is characteristic of the asthenic type, in whom the low flat diaphragm is associated with visceroptosis and an elongated centrally placed heart. On the other hand, in individuals of stocky build, the diaphragmatic vault is higher and the costo-phrenic sinus is deep. This is also true of stout persons in whom the excessive abdominal fat mechanically displaces the diaphragm upward. Statements concerning the height of the diaphragm have therefore only a limited value and inferences from it should be drawn guardedly. The right leaf of the diaphragm is usually slightly higher than the left; it should however be borne in mind, that gaseous distention of the stomach and colon which may arise from physiological causes, will result in an elevation of the left leaf of the diaphragm so that it may on occasion be as high as or higher than the right.

Having determined the position of the diaphragm the examiner is next concerned with its movement in respiration. This is most satisfactorily observed on the fluoroscopic screen. Normally the excursion of the diaphragm varies considerably with the individual type of breathing, depending on whether this is chiefly costal or abdominal. In long-chested persons, in whom the arch of the diaphragm is low and flattened, the respiratory movement may be minimal and consist only in a slight depression of its central tendinous portion, whereas the peripheral portion of the muscle remains almost immobile. In the short and deep-chested individuals the excursions are ample and with deep inspiration there is a widening and areation of the depths of the costo-phrenic sinus. Aside from these general characteristics of its respiratory move-

ment, considerable variation in the action of the diaphragm will be encountered which appears to be peculiar to the individual. Some patients find it impossible to contract the diaphragm voluntarily during the course of the examination and its motility cannot be studied until their attention is diverted from it. For this reason, an immobility of the diaphragm must not be assumed off hand and repeated effort must be made to instruct the patient in the proper manner of breathing. Jerky, irregular movements of the diaphragm are often caused by nervousness on the part of the patient.

The contour of the diaphragm is smooth and uniformly convex. During deep inspiration however, it is not uncommon for its mesial half to be separated from its lateral portion by a slight sulcus. This phenomenon is seen nearly always on the right side. In certain cases in deep inspiration this portion of the diaphragm is so elevated as to produce a localized projection upward into the chest which is apt to be confused with the prominence caused by a liver abscess or tumor. (Fig. 380.)



FIGURE 380

Prominence of mesial portion of the right diaphragm due to elevation of the central tendon in deep inspiration.

It is important to recognize the cause of this abnormality in the shape of the diaphragm because it is not due to organic disease nor is it even caused by adhesions. It is in all probability produced by an upward suction of the central tendinous portion of the diaphragm, through the inspiratory traction of the lung. It disappears entirely during expiration and has no clinical significance. The sulcus between the tendinous and muscular parts of the diaphragm is graphically brought to view in cases of pneumoperitoneum, in which the presence of air between the diaphragm and the liver permits a more comprehensive view of the former. (Fig. 396.)

ELEVATION OF THE DIAPHRAGM

The commonest causes of an abnormally high diaphragm are to be found in the abdominal cavity and they usually result in its elevation on both sides. These causes in brief, are gaseous or fluid distention of the abdominal cavity, intra-abdominal neoplasms and a coincident enlargement of the liver and spleen. Frequently an enlargement of the liver which affects both its right and left lobes will cause an upward displacement equally of both halves of the diaphragm.

We are however more especially concerned with a unilateral elevation of the diaphragm; a consideration of its causes presents for discussion a large number of pathological conditions in both the thoracic and abdominal cavities, on which the position and shape of the diaphragm may throw much light.

(1) Pleurisy without effusion commonly causes an upward displacement of the diaphragm. The elevation is usually of moderate degree and in most cases it is the only Roentgen evidence of the disease. Its cause is probably a reflex inhibition of the muscle due to the neighboring inflammatory process and it is therefore also present in lobar pneumonia and other acute inflammatory diseases of the lung which involve the pleura. In effect, the diaphragm does not contract in respiration and remains in the expiratory position. The immobility and elevation of the diaphragm are evidenced not alone throughout the acute stage of the disease but may persist for weeks thereafter.

The presence of a small amount of fluid during the course of an acute pleurisy, in no way alters the high position of the diaphragm. It is only when a larger amount of fluid has collected, that its weight will depress the diaphragm.

(2) SUBPHRENIC ABSCESS—is responsible for some of the most extreme upward displacements of the diaphragm. The presence on the right side of those organs which are most often responsible for suppuration in the subphrenic space namely, the appendix, pylorus, duodenum and liver, accounts for the great frequency of the disease on that side. It is evident that the degree of upward displacement of the

diaphragm will depend on the amount of fluid in the subphrenic space and also on the position of this fluid with respect to the diaphragm. Thus, a collection of pus on the posterior aspect of the liver, or a perinephritic abscess which has just extended to the subphrenic space, may in no wise influence the position of the diaphragm. For this reason, in a suspected case, a normally placed diaphragm should not be permitted to impugn the physical evidences of a subphrenic abscess. Aside from these exceptional circumstances, it is probable that in most cases, sooner or later the accumulation of pus will thrust the diaphragm upward to a noticeable degree. If this displacement is marked and if the causative factors warrant it, a Roentgen diagnosis of subphrenic abscess may be made with some assurance. Thus, the high diaphragm in figure 381 was the result of a collection of pus which followed an infected retrocolic appendix. When the upward displacement of the diaphragm is extreme the right lower lobe may suffer considerable compression. Under these circumstances the clinician may have some difficulty in defining the upper limit of liver dulness. On the Roentgen plate, however, the compressed lung may easily be distinguished from the elevated diaphragm.

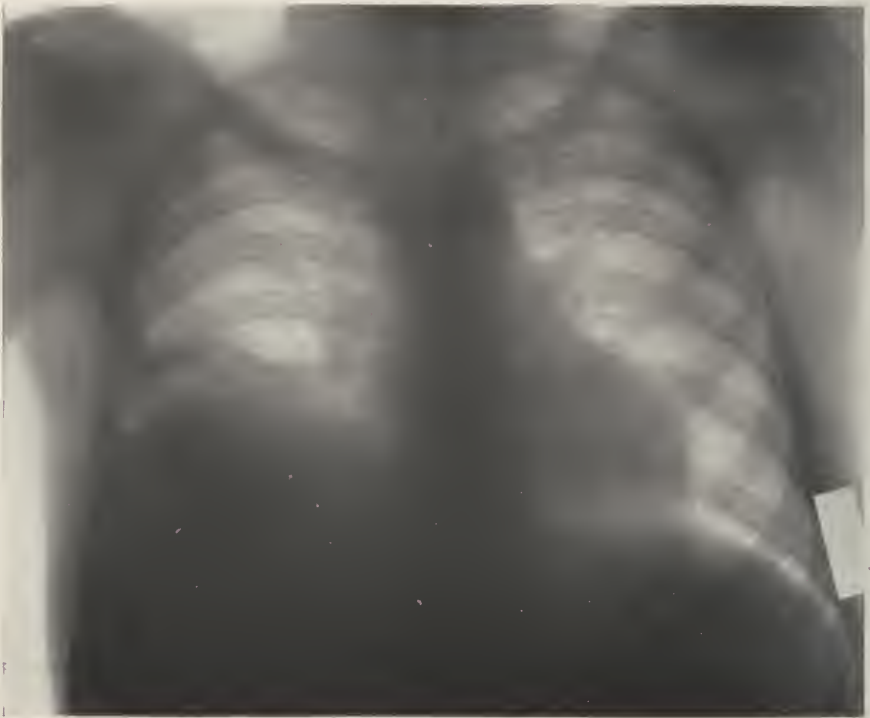


FIGURE 381

Subphrenic abscess. Diaphragm elevated and immobile. Fibrinous exudate on pleura at right base.

An inflammatory process below the diaphragm has the same effect on its mobility as one above it. The diaphragm is therefore fixed in respiration. It will be recalled that a pleurisy also brings about a slight elevation and immobility of the diaphragm. This fact has to be borne in mind because at least one-third of the cases of subphrenic abscess are complicated by either a dry pleurisy (fig. 381), or a pleurisy with effusion. It will therefore not always be an easy matter to decide whether the diaphragmatic elevation, especially when it is only slight, is due to a subphrenic abscess or to a pleural effusion or whether **both** these conditions, as frequently happens, coexist in the same case. It will be clear that in the diagnosis of subphrenic abscess, the observer can adhere to no arbitrary rule. An elevation of the diaphragm will be significant in proportion to its height; if the elevation is slight, its importance will lie in focussing the attention on the subphrenic space so that by repeated observation, both clinical and Roentgenological, an



FIGURE 382

Subphrenic abscess, left side. Stomach obscured by the fluid under left leaf of diaphragm. Pleural effusion on right side.

increase in the size of the abscess may be noted. Only in the extreme grades may the diagnosis be ventured with certainty from the Roentgen examination alone.

The shadow of a subphrenic abscess on the right side is superimposed on that of the liver from which it is indistinguishable. The diagnosis is therefore an inference drawn from an upward extension of the liver shadow. On the left side however, a collection of fluid in the sub-phrenic space may be directly visible owing to the presence in this situation of gas containing viscera which favor its visibility. The shadow of the fluid will then be seen in the region of the fundus of the stomach, which it may actually depress. An elevation of the diaphragm may therefore be entirely absent. This is graphically illustrated in figure 382. The left subphrenic abscess is indicated by the dense crescentic shadow which occupies the space normally filled by the gastric air bubble. It will be noted that the diaphragm is normal in position. At operation a collection of pus was found posteriorly, in the subphrenic space; its origin was an abscess of the spleen.

Attention has been drawn by observers to the occurrence of subphrenic effusions which undergo resolution without suppuration. The absorption of such effusions will no doubt explain the occasional reces-



FIGURE 383

Subphrenic infected hematoma following cholecystectomy.

sion of the diaphragm in cases in which subphrenic infection has been suspected. It is also worth while to call attention to the occasional rapid development of large non-purulent collections of fluid in the subphrenic space after operations on the liver and gall bladder. In these cases post-operative hemorrhage may occasion an enormous subphrenic hematoma, which, as in the case illustrated in figure 383, may extend up to the second rb. The rapid development of dulness on the right side of the chest and diminished breathing, with little or no temperature may easily lead to confusion with intrathoracic disease, an error which the Roentgen examination will at once set right. The hematoma may undergo prompt absorption with a return of the diaphragm to its normal position.

It is proper to add that an upward progression of the diaphragm under observation does not necessarily signify an exudate beneath it; a similar phenomenon may be observed in the rapid growth of a liver abscess and, like a subphrenic abscess, it may be associated with a pleural effusion.

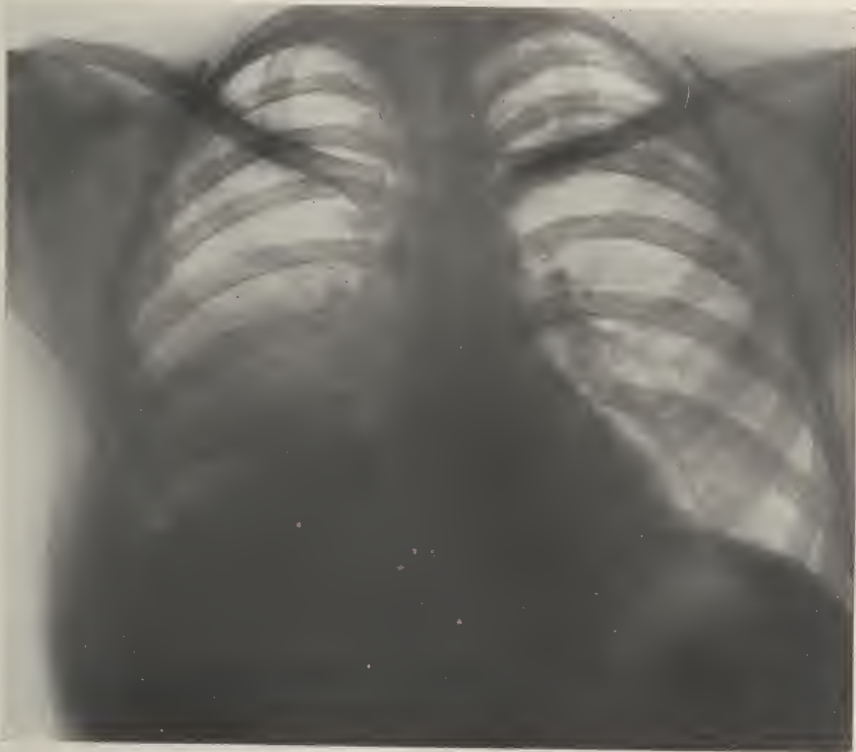


FIGURE 384

Subphrenic gas abscess secondary to perforation of duodenum. Early stage showing only a small amount of gas. Pleurisy at right base. See figure 385.

(3) SUBPHRENIC GAS ABSCESS. The difficulties in the diagnosis of ordinary subphrenic abscess no longer exist when in addition to pus, gas obtains access to the subphrenic space. The presence of the latter causes the fluid, which is otherwise indistinguishable from the liver shadow, to assume a horizontal level, above which the bubble of gas is sharply outlined by the curve of the diaphragm. In order to demonstrate this fluid level, the examination must of course be made in the upright position. The gradual increase in the effusion can be gauged by an extension of the fluid level and also by the amount of gas. Thus in figure 384 a small collection of gas was found beneath the diaphragm a few days after an operation on the common bile duct, during which the duodenum was perforated. A few days later the gas and fluid had increased so that they extended entirely across the subphrenic space on the right side. (Fig. 385.) In the case illustrated in figure 386 the gas became evident only when the abscess had reached a great size and had extended up to the level of the second rib.



FIGURE 385

Subphrenic gas abscess, showing increase in size of exudate. Plate made two days after figure 384. Dorsal view.



FIGURE 386

Large subphrenic abscess, with gas.

The discovery of gas in these cases has considerable practical importance, as it usually, if not always indicates a perforation of the stomach or intestine, which may require more radical treatment than simple evacuation of the abscess.

The term "subphrenic pyopneumothorax" which is occasionally applied to this condition reflects the uncertainty which may attend its physical diagnosis. When large amounts of gas are present in the abscess, the physical signs are very similar to those which result from an accumulation of air and fluid in the chest and it is apparent, from the definite nature of the Roentgen findings, that these are calculated at once to localize the disease in the abdomen rather than in the chest.

(4) An elevation of the diaphragm may be caused by new-growths, gummata and abscesses of the liver. Although occasionally only a segment of the diaphragm may be elevated, in the majority of cases the dome of the diaphragm is uniformly raised on one side and its contour is not significantly altered. The observer must again be cautioned to disregard the prominence of the mesial half of the right diaphragm, consisting of its central tendinous part, which may become separated

from the liver as a boss-like protuberance, as it has no pathological significance. Only on the left side has such a deformity of the diaphragm any significance and here it may be due to localized disease of the liver or spleen. Thus in figure 387 the prominence of the mesial half of the left diaphragm was caused by a large echinococcus cyst of the spleen. On fluoroscopic examination this was seen to involve only its anterior half, the posterior portion remaining at the normal level. At operation no abnormality was found in the liver to account for the prominence of the right diaphragm.



FIGURE 387

Elevation of the left diaphragm by an echinococcus cyst of the spleen.

(5) EVENTRATION OF THE DIAPHRAGM. It may truthfully be stated that this rare and interesting disease was practically unknown and its clinical diagnosis was almost impossible before the era of Roentgen examination. It is more correctly termed diaphragmatic atrophy as this is the essential lesion and the eventration is but its result. Its etiology is shrouded in obscurity. Variouslly ascribed to a congenital atrophy or dystrophy of the diaphragm or to a neuro-muscular degeneration following a phrenic neuritis or polio-myelitis, in any case the muscle fibres undergo a degeneration which may terminate in a fatty or

fibrous replacement. Tone and power of contraction are lost and the diaphragm, yielding to the pressure of the stomach and intestines, is displaced progressively upward into the thorax together with the abdominal viscera.

Closer study of this condition is warranted in spite of its rarity because the symptoms which it may produce are usually confused with those of other, more common diseases. For example, one group of symptoms which result probably from a sudden upward pressure on the already displaced diaphragm, consists in periodic attacks of dyspnoea and sharp pain in the left side of the chest. When such patients are examined, the tympanitic note over the chest due to stomach and gut, together with the marked cardiac displacement may readily betray the examiner into an erroneous diagnosis of spontaneous pneumothorax. The correct diagnosis is made at a glance by the plate or fluoroscopic screen. (Figs. 388 and 389.) The diaphragm is here seen as a sharp linear band high in the chest, which may extend as far as the second rib, bounding the distended stomach and intestinal coils beneath it. Invariably the heart is pushed completely to the right side.

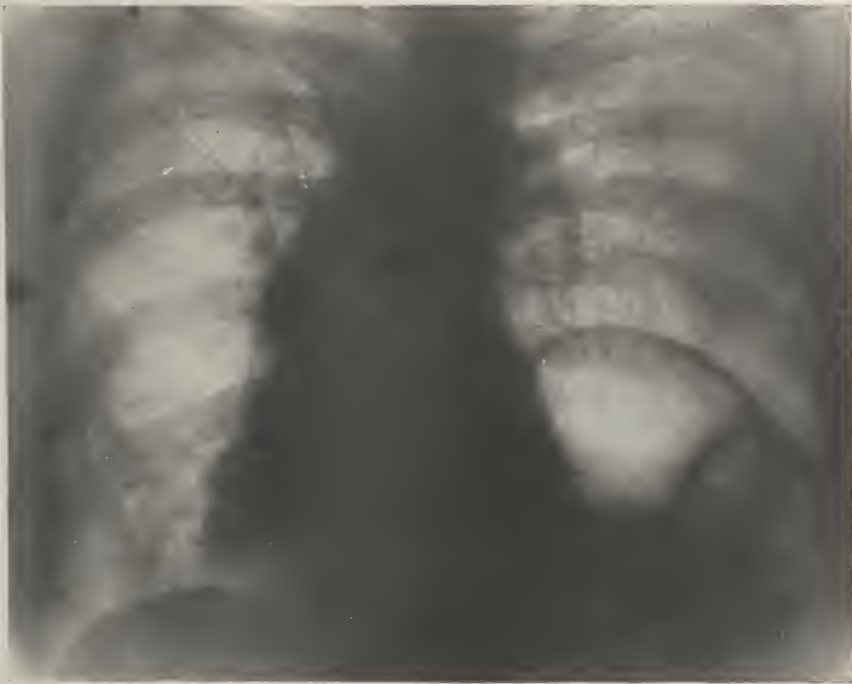


FIGURE 388

Eventration of the diaphragm, left side. Dextro-cardia.



FIGURE 389

Eventration of the diaphragm showing distended stomach and colon. Heart completely displaced into the right chest.

It need hardly be pointed out, that in the diagnostic aspiration of the chest in these cases, in which pleurisy or pneumothorax may be suspected, there is a very real danger of perforating the distended stomach or intestines.

Again, the patient may present symptoms due to intestinal obstruction as a result of atony and dilatation of the loops of intestine in the eventration. Thus, the eventration on the right side shown in figure 390 was associated with an enormous dilatation of the colon, which was responsible for most of the symptoms. Its distended coils are seen just below the diaphragm. At operation the diaphragm was found intact but atrophied. In right sided eventration the liver is displaced downward and its place is occupied by coils of intestine. The protean symptomatology of this interesting disease is illustrated by those cases in which the extreme displacement of the heart gives rise to cardio-vascu-



FIGURE 390

Eventration of diaphragm, right side. Diaphragm at level of second rib.
Dilatation of colon. Ptosis of liver.

lar symptoms. In no other disease is the heart so completely thrust into the right chest and this condition is not uncommonly mistaken for congenital dextro-cardia. So true is this that a diagnosis of dextro-cardia can never be regarded as firmly established until an eventration of the diaphragm has been excluded by Roentgen examination.

On fluoroscopic examination the convex linear shadow of the diaphragm shows no active respiratory movement. In certain instances a paradoxical movement upward during inspiration may be observed; this may be conceived as a passive displacement due to the contraction of the normal half of the diaphragm which forces the intestines upward. The distinction between eventration and diaphragmatic hernia is usually not possible by means of the Roentgen Ray. Occasionally a study of the intestinal contents of the hernia after the ingestion of bismuth may throw some light on this condition, especially in recent



FIGURE 391

Hernia of the stomach through a small bullet wound of the diaphragm. Stomach filled with barium.

cases of hernia following trauma such as are due to bullet wounds. (Fig. 391.) In the more extensive cases and especially in the rare instances of congenital hernia, the Roentgen appearance of the two conditions may be identical. This is unfortunate as the differentiation of these two disorders has a therapeutic importance. Diaphragmatic hernia may in certain cases be relieved by operation whereas we have no such remedy for the relief of an atrophic diaphragm. It is possible that the injection of gas into the peritoneal cavity may become a useful method in the differentiation of eventration from diaphragmatic hernia. In a recent case which we studied, after the production of a pneumoperitoneum, the fundus of the stomach was caused to fall away from the atrophic diaphragm above it, proving conclusively that the diaphragm was intact and that the condition was an eventration. (Fig. 392.)

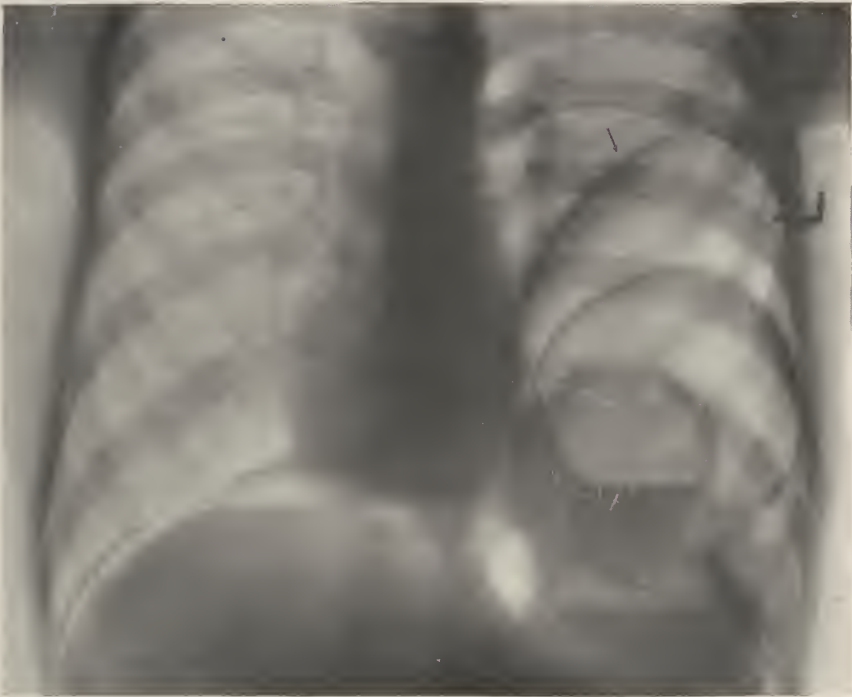


FIGURE 392

Eventration of the diaphragm, distinguished from diaphragmatic hernia by means of pneumoperitoneum. The diaphragm, indicated by arrow is seen to be intact and is separated from the stomach and colon below it by the air in the peritoneum.

A sharp distinction must be drawn between the extreme upward displacement of the diaphragm which is so characteristic of eventration, and the moderate elevation which is not uncommonly associated with a chronic dilatation of the fundus of the stomach. The gastro-enterologist is apt to encounter these patients in his practice and the discovery in them of a high diaphragm due to a gaseous distention of the stomach occasionally supplies a clue to the origin of their symptoms. The diaphragm is here not atrophic and it functions normally in respiration. (Fig. 393.)

(6) PHRENIC PARALYSIS. A lesion of the phrenic nerve results in an immobility of the affected side of the diaphragm which assumes the expiratory position. Aside from such causes as true neuritis and polio-myelitis, the Roentgen examination discloses other agencies for this condition as tumors of the lungs and mediastinum and the chronic mediastinitis of tuberculosis. In figure 62 the high, par-

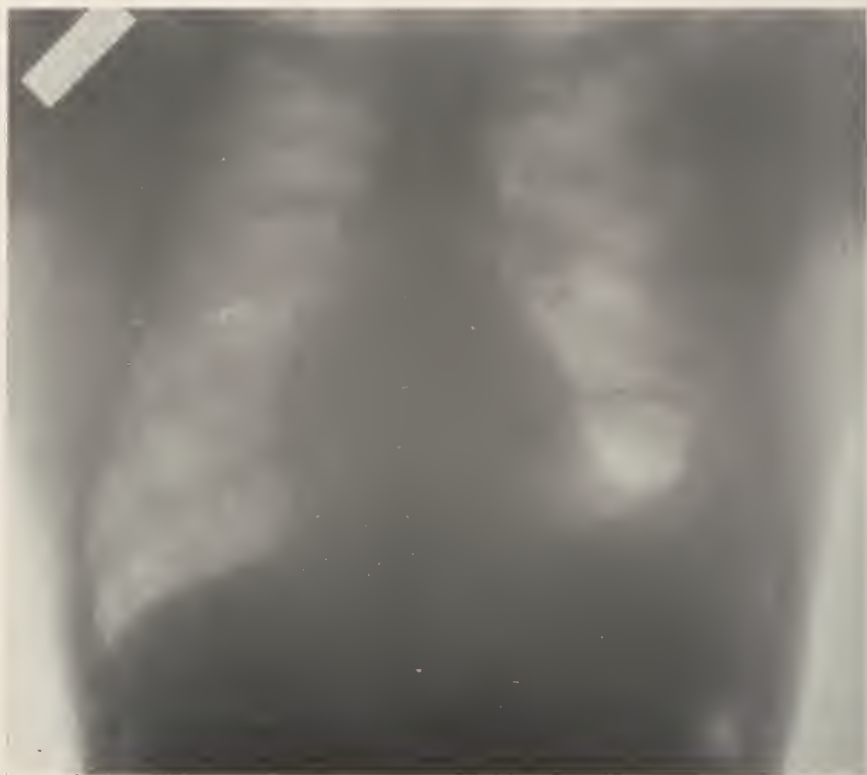


FIGURE 393

Chronic, atonic dilatation of stomach with elevation of the diaphragm.

alytic diaphragm is probably due to pressure on the phrenic nerve by the mediastinal adhesions of tuberculosis. There is further evidence of a chronic mediastinitis in the marked displacement of the trachea, heart and aorta toward the right.

DEPRESSION OF THE DIAPHRAGM

An abnormally low position of the diaphragm is much less common than its elevation and it results always from intrathoracic causes. The elastic pull of the lungs constantly exerts its force in opposition to any causes within the abdomen which would tend to displace the diaphragm downward. It is therefore only when this force is compromised that the diaphragm may suffer a marked depression. This condition is found in its extreme form in cases of pneumothorax, especially when the air within the pleural cavity is under great tension. The diaphragm may then be flattened or it may even take an oblique course downward

from its fixed point near the spine to the chest wall. (Fig. 288.) When the pneumothorax is associated with fluid in the pleural cavity, the weight of the fluid may even cause the diaphragm to become convex downward; this can be observed only on the left side where the diaphragm is outlined against the air in the stomach.

PNEUMOPERITONEUM

When gas gains access to the peritoneal cavity, even in very small quantities, one of its notable effects is to cause a separation of the liver from the diaphragm. This interposition of gas between the diaphragm and the upper surface of the liver is such a characteristic feature of the Roentgen plate that it may be regarded as positive evidence of free air or other gas in the peritoneal cavity. When larger amounts of gas are present the spleen and other organs may be separated from the abdominal wall and they may be visible in their entirety. (Fig. 394.) We are here, however, only concerned with right-sided pneumoperitoneum, whose recognition may throw light on certain thoracic or intraabdominal diseases. Thus, in a patient who



FIGURE 394

Induced pneumoperitoneum showing outline of diaphragm above and liver and spleen below.

presented definite symptoms of gastric ulcer, following a violent paroxysm of pain and other symptoms which suggested the likelihood of a perforation, the Roentgen examination revealed the pneumoperitoneum seen in figure 395. In conformity with this finding a large



FIGURE 395

Pneumoperitoneum, right side, secondary to perforated gastric ulcer.

perforation in a gastric ulcer was found. The Roentgen examination is particularly adapted to the demonstration of peritoneal gas in cases of slow perforation of an ulcer in which the symptoms are less fulminating and in which it is therefore possible without danger to the patient, to perform the examination in the upright position.

Gas introduced into the abdominal cavity from without either for diagnostic purposes or accidentally will produce an identical appearance. In figure 396 the subphrenic collection of gas followed an unsuccessful attempt at artificial pneumothorax. The needle was inserted too low and, penetrating the costophrenic sinus, entered the subphrenic space into which the gas was mistakenly injected.



FIGURE 396

Pneumoperitoneum. Diaphragm separated from liver by air introduced during performance of artificial pneumothorax.

Pneumoperitoneum must not be confounded with the condition of hepatoptosis in which a similar though not identical phenomenon may at times be observed. The ptosis and forward rotation of the liver may permit the transverse colon to become interposed between it and the diaphragm. The Roentgen plate will then show a collection of gas between the diaphragm and the liver; in this case however, the gas is enclosed within the intestinal walls, whose haustrations are distinctly visible. (Fig. 397.) In the recumbent position the liver resumes its usual position.



FIGURE 397

Ptosis of the liver. Transverse colon, showing haustrations, interposed between it and the diaphragm.

Among the rarer causes of a depression of the diaphragm we may mention stenosis of the larynx and a unilateral bronchostenosis. The diaphragm is depressed only when the obstruction is of moderate grade, especially when the exit of air from the lungs is most interfered with, so that the latter are over-distended. On the other hand with a high degree of stenosis of the larynx or the complete closure of a main bronchus, the lung becomes airless and the diaphragm is elevated. This phenomenon is of value in the diagnosis of tumors or non-opaque foreign bodies in a bronchus.

DIAPHRAGMATIC ADHESIONS

Perhaps the most common abnormality exhibited by the diaphragm is the pleuro-phrenic adhesions which result from inflammation of the pleura. They are most often the sequel of pulmonary tuberculosis and are found in association both with recent and acute disease of the lungs and with the older and healed lesions. The effects of adhesions are evidenced either by a change in the contour of the diaphragm or a restriction of its movement or in both of these respects. Thus, the smallest adhesions may not be visible as such but may cause only a slight lagging of the diaphragm on deep inspiration. Especial attention must be focussed on the mesial portion of the diaphragm as the lagging is often confined to this region, whereas the lateral two-thirds move freely.

**FIGURE 398**

Pointed diaphragmatic adhesion. Chronic pulmonary tuberculosis.

A common site for the diaphragmatic adhesions of healed tuberculosis is in the costophrenic sinus which may be obliterated. Nothing is more common than the faint shadow which obscures the contour of the diaphragm as it dips down in the lower axilla. On fluoroscopic examination this region fails to become aerated as it normally should in deep inspiration. In other cases the diaphragm may be raised up in a tent-like prominence. (Fig. 398.) A series of adhesions may give the diaphragm a serrated contour. (Fig. 31.) Finally, its outline may be hazy consequent on a total obliteration of the pleural complementary space. This is especially marked in the cases of fibroid tuberculosis with excessive thickening of the pleura, in which the diaphragm may be entirely obscured by pleural adhesions and thickening. (Fig. 64a.)



FIGURE 399

Distortion and fixation of diaphragm in chronic empyema.

Aside from pulmonary tuberculosis, other thoracic diseases may bring about striking deformities of the diaphragm. The pleural thickening which is such a common sequel of empyemata often exerts its maximum effects on the axillary portion of the diaphragm, which it fixes in an elevated position in this region. The diaphragm may therefore rise obliquely from the spine toward the axilla and thus endow the lower chest with an unusual conformation. (Fig. 399.) In other cases as a result of this fixation of the diaphragm, it no longer glides over the liver during respiration but it is on the contrary drawn taut in a straight line, which may closely resemble a fluid level. (Fig. 400.) On the other hand, the central tendinous part of the diaphragm may in cases of mediastinal disease, bear the brunt of the adhesive process. Thus the chronic mediastinitis associated with aneurysms and mediastinal



FIGURE 400

Fixation of the left leaf of the diaphragm in its peripheral part with loss of its normal convexity.

tumors may, through the resulting adhesions, exert an upward traction on this portion of the diaphragm which acquires the unusual conformation illustrated in figure 309.

Diaphragmatic adhesions are occasionally responsible for puzzling Roentgen pictures which may be difficult to interpret unless the previous history is available. For example, a middle aged man who had recently been operated on for a gastric ulcer, which was possibly malignant, continued to suffer upper abdominal pain and became emaciated. During the gastro-intestinal Roentgen examination a prominence of the mesial half of the right diaphragm was noted. (Fig. 401.) Naturally, suspicion of a metastatic tumor of the upper surface of the liver arose. The patient had been in the hospital two years before with a right lower lobe pneumonia which persisted for some months, the Roentgen plate



FIGURE 401

Elevation and prominence of mesial part of right diaphragm. Secondary to pneumonia three years before. See figure 402.

of which was fortunately available. At that time already (fig. 402) the diaphragm was elevated and partially adherent and the extreme condition found on the subsequent examination was thus foreshadowed.



FIGURE 402

Same case as figure 401 showing pneumonia of the right lower lobe two years before, with beginning elevation of the diaphragm by adhesions.

ABNORMALITIES OF DIAPHRAGMATIC MOVEMENT

We have already mentioned the inhibition of the diaphragm, usually unilateral, which is a manifestation of an inflammatory process in its vicinity. In its relation to the diagnosis of pulmonary tuberculosis, rigidity of the diaphragm has also been considered. In this connection it may be added that in the more chronic forms of the disease and especially in arrested and cured cases, the function of the diaphragm, provided there are no adhesions, does not appear to be impaired.

Various abnormalities in the respiratory movement of the diaphragm are occasionally to be seen on the fluoroscopic screen. In cases of pneumothorax and seropneumothorax a paradoxical movement may be visible. This may also be seen at the very beginning of inspiration, when there are extensive pleuro-phrenic adhesions. Adhesions

between the pericardium and the diaphragmatic pleura may exert a systolic pull on the diaphragm which becomes most evident on deep breathing. When this condition is complicated by an obliteration of the pericardial cavity, this phenomenon may express itself clinically as a Broadbent sign. (See figure 99.)

In bulbar palsy the diaphragm may be immobile on both sides and assume a high paralytic position. A unilateral paralysis of the phrenic nerve results in a high immobile diaphragm on the affected side which is best brought out in the recumbent position. Owing to the continuance of costal breathing the diaphragm may be displaced further upward during inspiration.

An increase in the movement of the diaphragm is less often observed than its inhibition. In some cases of emphysema in which the inspiratory movement is restricted by the rigidity of the chest wall, there may be compensatory over-action of the diaphragm whose contour becomes flattened to a degree never seen in other conditions. Finally it should be noted that a marked overaction of one half of the diaphragm is at once suggestive of disease on the opposite side, particularly obstruction within a bronchus.

SECTION IX

Surgical Diseases of the Chest

CHAPTER XXVIII

Surgical Diseases of the Chest

Roentgenology of the chest has nowhere received a more practical or valuable application than in the study of surgical diseases of the lungs and pleura. It has become indispensable not only for the diagnosis and localization of intrathoracic diseases but also for the accurate oversight of their postoperative course and the numerous complications which are apt to beset a patient whose chest has been opened.

We will first discuss some of the surgical aspects of empyema which is by far the commonest thoracic disease that is surgically treated. We have in a previous chapter considered certain unusual features of empyema in the diagnosis of which, previous to operation, the Roentgen examination plays an outstanding part. After thoracotomy however, the labors of the Roentgenologist are by no means ended; residual collections of pus, which are at times situated in remote recesses of the chest, often wait upon the Roentgen plate for their accurate and timely localization. Thus it is of special help in the early recognition of residual apical empyemata, (figs. 255 and 257) which otherwise are apt to remain undiscovered until they are spontaneously emptied through a bronchus. A second thoracotomy is usually required for their evacuation as it is not practicable to drain them through a low thoracotomy along a tortuous sinus.

The mesial empyemata which occasionally develop after operation are of great interest from a surgical standpoint. In our experience they follow effusions which were originally encapsulated in the lateral half of the chest, its mesial portion being protected by adhesions. The secondary empyema is the result of a separation of these adhesions and an infection of the mesial part of the pleural cavity during or after operation. As the fluid is here walled off by adherent lung it may exert pressure on the mediastinum and occasion some dyspnoea.

The mesial empyema shown in figure 254 was clearly a consequence of infection during operation; the larger collection of pus and air in figure 403 was formed some time after operation and gave rise to considerable respiratory difficulty. In each case prompt recovery followed localization of the pus by the Roentgen Ray and its evacuation through an intercostal incision made close to the sternum. The ease of the Roentgen demonstration of these mesial empyemata stands in marked

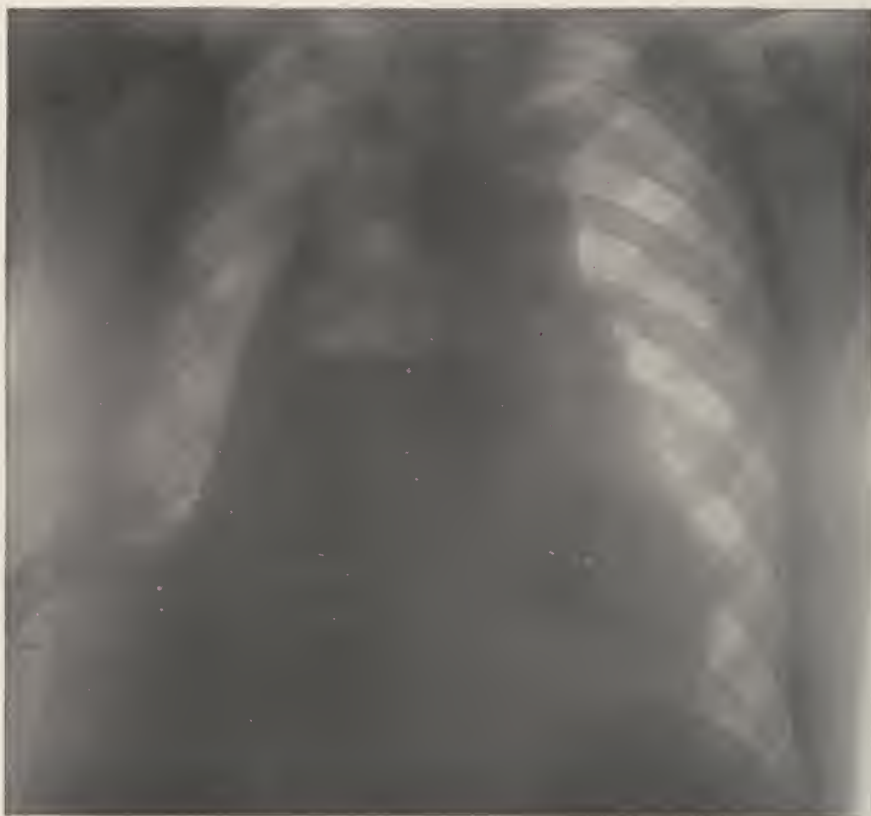


FIGURE 403

Postoperative empyema with air, in mesial half of chest. Original empyema completely drained.

contrast to the difficulty of their recognition clinically. The altered physical conditions which exist in the chest after it has been opened and especially the presence of air in the pleural cavity, enormously enhance the difficulties of the clinical examination. Moreover, exploratory aspiration near the border of the sternum, without the help furnished by the Roentgen plate, is not to be lightly undertaken, whereas under its guidance the procedure is absolutely safe.

In cases of empyema, after all the pus has been drained from the pleural cavity there remains an infected pneumothorax of variable size, depending on the degree of pulmonary collapse. The obliteration of this pneumothorax depends ultimately on the ability of the collapsed lung to expand and to fill up the dead space. A persistence of the purulent discharge sooner or later prompts an investigation of the mobility of the lung and of the size, shape and situation of the pneumothorax or sinus.

The Roentgen findings associated with a persistent empyema sinus are usually as easy to interpret as they are instructive. In the simpler cases there is found an area of pneumothorax of variable extent in the axillary region, which is mesially limited by the partly expanded lung. The lung itself may be well aerated and free of adhesions (fig. 404) or



FIGURE 404

Healing empyema, left side. Lung almost expanded leaving a narrow zone of pneumothorax. Thickening of parietal pleura and also visceral pleura outlining margin of lung.

it may be thickened and covered by a dense exudate which binds it firmly to the chest wall. There is usually also a homogeneous shadow in the lateral portion of the chest which is due to a thickening of the parietal pleura. (Fig. 405.) Depending on the efficiency of the drainage more or less fluid which exhibits a horizontal level, will be found in the chest. A characteristic picture of persisting empyema at this stage is seen in figure 406.



FIGURE 405

Extrapleural compression of chest for chronic empyema. Extensive rib resections. Note dense shadow of collapsed lung along heart border and large pneumothorax. (x) indicates characteristic shadow of thick parietal pleura.



FIGURE 406

Persisting empyema sinus. Thickening of parietal pleura. Retention of pus in lateral half of chest.

In the more complicated cases the pneumothorax is irregular and may consist of a tortuous channel containing one or more pockets of infection. The unaided plate is of little avail to unravel the intricacies of these sinuses unless it is supplemented by an injection of the fistulous tract with some opaque substance, such as bismuth. The resulting pictures are very instructive as they portray with the minutest detail the configuration of the pneumothorax. (Figs. 407 and 408.)



FIGURE 407

Chronic bilocular empyema sinus outlined by injected bismuth.

A most important factor concerned in a persistence of the pneumothorax is the rigidity of the collapsed lung. This will determine to a great extent, the operative procedure which may later be undertaken to bring about a cure. The Roentgen plate often indicates by the density of its shadow, whether the lung is free or is bound down by a thickened visceral pleura or fixed by adhesions to the chest wall. Thus in figure 406 the retracted lung is well aerated and presumably not



FIGURE 408

Chronic empyema sinus. Cavity in pleura filled with injected bismuth. Diaphragm drawn upward by adhesions.

firmly bound by adhesions; as the fluid was absorbed, it rapidly expanded and obliterated the pneumothorax. In figure 404 the mobile lung has nearly unfolded itself so that only a narrow zone of pneumothorax remains between it and the thickened parietal pleura.

The firmness of the adhesions which bind the lung can be more directly ascertained by fluoroscopic examination during deep inspiration or forced expiration. The ability of the lung to expand may thus be determined; if it is absolutely fixed it is unlikely that the minor palliative operations will suffice for a cure and major procedures may have to be resorted to. In these cases there is usually found an extreme

collapse of the lung which may be compressed against the spine and covered by a much thickened pleura. Commonly there is also seen a thickened parietal pleura, (fig. 405,) which, being essentially a pyogenic membrane, is the origin of the purulent discharge and contributes much to a persistence of the disease. The shadow of the parietal pleura offers some difficulty in interpretation. It is not evenly diffused over the chest, as might be expected but is most intense in the axillary region and it is sharply delimited from the pneumothorax mesial to it. The width of this shadow is not an exact measure of the pleural thickening as its peculiar appearance is merely an optical phenomenon produced by the curvature of the chest wall.

In the various surgical procedures which are undertaken to remedy the conditions we have just described, the value of the constant oversight afforded by the Roentgen Ray cannot be overestimated. It will suffice to illustrate the end results of some of these operations, such as decortication of the lung, (fig. 409,) and the newer thoracoplastic operation of Sauerbruch, (fig. 405,) in order to indicate the wide field of usefulness of the Roentgen examination.



FIGURE 409

Thoracic deformity resulting from decortication of the lung for chronic empyema. Extensive rib resections.

Although diseases of the lung are much less amenable to surgical treatment than are pleural diseases, the field of pulmonary surgery is being rapidly extended. Pulmonary suppuration, new growths and tuberculosis, to mention the more common ones, are the diseases which, with increasing frequency, are referred to the surgeon. Here also, as in the case of pleural effusions, the Roentgen examination is indispensable, both for diagnosis and in the course of treatment.

Especially in respect to lung abscess is there need for accurate localization of the diseased area of the lung. Whether the intention of the surgeon is merely to drain an abscess cavity or to extirpate a whole lobe, it is necessary to know which lobe or lobes are involved in order that the incision be well placed. In most cases this is readily determined on the Roentgen plate, especially if the shadow of the diseased area occupies the apex or base of the lung. When however, the infiltration occupies the middle third of the lung, it may be impossible to decide whether the process involves one lobe or the adjacent portions of three lobes. The reason for this is the overlapping of the three lobes in the middle third of the pulmonary field which causes their shadows to be superimposed. The difficulty is especially great in the case of the middle lobe. When it is involved alone its appearance is characteristic, (fig. 158,) but when the adjacent portions of the other lobes are consolidated, its outline can no longer be distinguished. (Fig. 179.) In these cases, dependence must be placed on the physical signs, whose situation on the anterior or posterior aspect of the chest will be of great help in interpreting the Roentgen shadows. For the localization of cavities, especially to determine their depth in the lung, nothing is more useful than the fluoroscopic examination. A cavity nearer the anterior chest wall will appear smaller but more sharply outlined when the rays traverse the chest in the dorso-ventral direction and vice versa.

Another common query addressed to the Roentgenologist before operation has to do with the presence or absence of pleural adhesions, which will affect the operative technique. Adhesions are frequently present, especially when the abscess is near the surface of the lung and has caused an inflammation of the pleura. They may be inferred from an increased density of the shadows and also from a retraction of the ribs. The introduction of a small amount of air into the pleural cavity with a partial collapse of the lung may be of service to visualize these adhesions and gauge their firmness. It is not possible to more than touch on the numerous occasions when the Roentgen Ray is invoked after lobectomy for lung abscess. During this eventful period when such serious complications as mediastinal flutter, tension pneumothorax and putrid empyema momentarily threaten the life of the patient, we have in the fluoroscopic screen and the plate a rapid and certain method

for their recognition. Finally, the Roentgen Ray cannot be dispensed with when we wish to judge the efficacy of the various methods employed for the extrapleural compression of the lung for suppurative or tuberculous disease. (Figs. 410 and 411.)



FIGURE 410

Extrapleural compression of residual lung abscess after partial lobectomy. The shadow in upper axillary region represents paraffin oil injected extrapleurally.



FIGURE 411

Same case as figure 410. Extrapleural compression of lung by inflated rubber bag.

SECTION X

Diseases and Abnormalities of the Chest Wall

CHAPTER XXIX

Diseases and Abnormalities of the Chest Wall

The soft tissues and the bones of the thorax occupy a prominent place on the Roentgen plate where they contribute notably to the shadows which traverse the chest. Under the influence of intrathoracic disease the chest wall is often secondarily affected leading to various deformities of its bony parts to which we have already alluded.

Study of the thoracic wall has however an interest for the Roentgenologist which transcends any possible bearing on disease of the intrathoracic organs. We must bear in mind that when the ribs, spine or the soft tissues of the chest are the seat of disease the abnormal shadows which they cast will be projected on those of the heart, lungs and pleura. Usually no confusion will arise as the shadows will bear intrinsic evidence of their origin. On the other hand, their connection with the thoracic wall will not always be obvious at a glance so that the exam-



FIGURE 412

Maldevelopment of right first rib, which articulates with the second rib.

iner will at times be at a loss to decide whether they are due to abnormal shadows within or without the chest. Whenever atypical shadows are encountered, it is advisable therefore to inspect or palpate the chest wall in order to discover a tumefaction of the soft parts or of the bones and in doubtful cases to resort to stereoscopic and fluoroscopic examination in various positions.

Of the abnormal shadows which arise from the thoracic wall, the most striking and numerous are seen in the bony thorax, especially the ribs. A considerable number of diseases and congenital abnormalities may affect the ribs, especially in the upper aperture of the chest.

The most common of these are the various forms of cervical ribs; in most cases they are symptomless and are found incidentally to examination of the lung. There are, besides true cervical ribs, numerous abnormalities of the upper aperture of the chest consisting of mal-development of the first rib, (fig. 412.) partial or complete fusion of the first and second ribs, (fig. 413,) or an aplasia of the upper chest



FIGURE 413

Fusion of left first and second ribs. Pulmonary tuberculosis.

with a resulting deformity and asymmetry. (Fig. 414.). Although these bony changes are at once apparent on the Roentgen plate and although their influence on the pulmonary physical signs may be not inconsiderable, they are not always obvious to the physical examiner. It is therefore not uncommon for patients with atypical upper ribs to be suspected of tuberculosis because of the apical dulness which they may produce. It is easily seen how the percussion note will be altered at the right apex in figure 413 by the fused first two ribs, which almost



FIGURE 414

Aplasia of the third and fourth ribs on the right side with depression of that portion of the chest.

obscure it. Minor changes, such as bifid sternal ends of the ribs, (fig. 415,) are occasionally seen and have mainly an interest for the anatomist. At times they produce a local prominence which is mistaken for a bony tumor.

Tumors of the ribs possess a great interest for the Roentgenologist because the shadows which they cast may overlie the pulmonary fields. On cursory examination, these shadows may closely resemble those of pulmonary or pleural diseases and one is readily betrayed into a wrong diagnosis. This is particularly true of the primary sarcomata of the ribs, in which their bony substance is destroyed and replaced by



FIGURE 415
Bifid sternal end of the left third rib.



FIGURE 416
Oblique and Dorso-ventral view of sarcoma of seventh right rib. Shadow in latter position suggests pleural disease.

a soft tumor mass. Their shadows are of moderate density and are well circumscribed. Usually, the healthy portion of the rib will be seen to terminate in this shadow and no evidence of it will be seen within the tumor mass. The similarity of such rib sarcomata to a localized pleural effusion is shown in figure 416. Although the dorso-ventral examination may leave us in doubt as to the nature of this shadow, any uncertainty is resolved by an oblique examination in which its origin from the rib is disclosed. This form of bone sarcoma is often multiple and such circumscribed shadows may then be seen in more than one rib. Thus in figure 417 tumors are seen in the right apex and at the left base



FIGURE 417

Multiple rib sarcomata. Shadows at right apex and left base.

arising from the ribs in these situations. In this case a bony reticulum is visible throughout the tumor.

In another form of rib sarcoma, the tumor may be of the infiltrative type and may spread far beyond the limits of the rib. The abnormal shadow may then be extensive and its bony origin may easily be overlooked. Thus, in figure 418, there is a shadow in the right lower lobe region which by itself is not distinguishable from one arising in the lung or pleura. It appears to have no anatomical relation to a rib, yet a close inspection of the seventh rib shows an area of bone absorption strongly suggestive of a new-growth. At operation, a large sar-



FIGURE 418

Sarcoma of the seventh rib on right side extensively invading the soft tissues of the chest. Shadow resembles a pneumonic consolidation. Note rarefaction of seventh rib. Dorsal view. See figure 419.

coma, arising from this rib and infiltrating the soft tissues of the chest wall was found and excised. The pleural cavity was not invaded. The appearance of the chest after excision of the ribs and tumor, is seen in figure 419 in which the abnormal shadow is no longer present. In this case the diagnosis received no aid from the physical examination as the tumor was barely palpable. It gave rise however, to a severe intercostal neuritis.

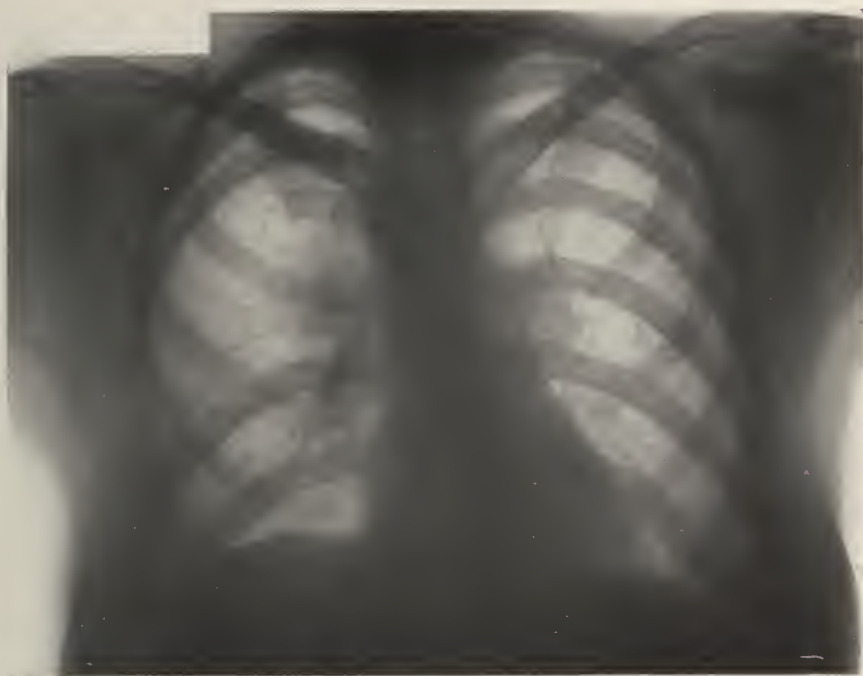


FIGURE 419

Same case as figure 418 after excision of tumor, showing that the shadow was extrapulmonary. Ventral view.

True bony tumors of the ribs may cast very dense bizarre shadows over the pulmonary fields, as in figure 420, which illustrates a large osteoma.

Among the rarer diseases which affect the ribs, may be mentioned multiple myeloma. The pathological process results in multiple areas of rarefaction and absorption of the bony structure, so that the ribs have a moth-eaten appearance. The bones of the shoulder girdle may



FIGURE 420

Osteoma of sternal end of second left rib.

also be affected and may appear cystic. (Fig. 421.) The Roentgenologist is often the first to recognize these cases because of the frequent pathological fractures of the ribs and spine which usually prompt an examination of the chest.

Next to the ribs, disease of the vertebrae as a source of abnormal thoracic shadows will engage the attention of the Roentgenologist. Sarcomata arising from the spine may form large tumor masses whose shadows lead to confusion in diagnosis, especially if a swelling is not externally visible. The difficulties of diagnosis are strikingly shown in



FIGURE 421

Multiple myeloma. Rarefaction of ribs, spine and bones of shoulder girdle.

the case illustrated in figure 422. The large left apical shadow was assumed to be due to a pulmonary or mediastinal tumor which it closely resembles, until severe pains in the neck led to a Roentgen examination of the cervical spine. Erosion of several vertebrae and the later development of a pathological rib fracture pointed to the probable bony origin of the tumors. At autopsy multiple sarcomata of the ribs and spine were found. The shadow was cast by a large tumor of the cervical spine and contiguous rib arches which, extrapleurally, projected into the thoracic cavity.



FIGURE 422

Large sarcoma of cervical spine and adjacent ribs, resembling a mediastinal tumor.

Tuberculous abscesses of the vertebrae are at times responsible for an unusual Roentgen picture. They are oftenest seen in the lower thoracic or in the abdominal cavities and produce a fusiform or globular shadow situated about the spine. As a rule they are partly obscured by the heart and cannot be seen in their entirety. In figure 423 the

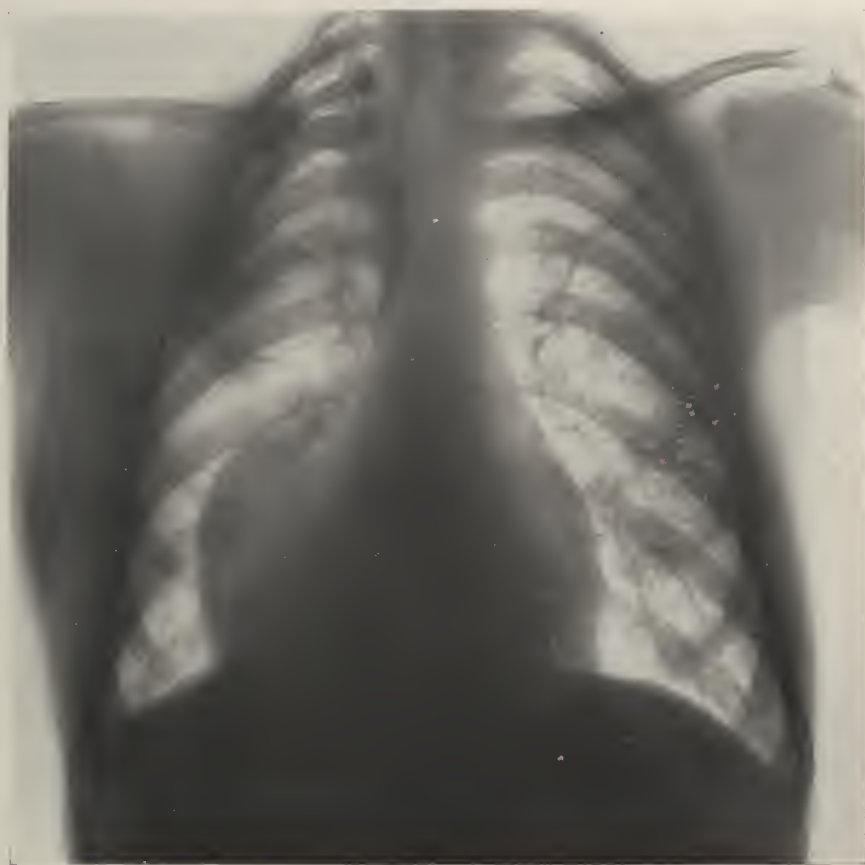


FIGURE 423

Large Pott's abscess in lower dorsal region extending into right lung field. On left side, outline of abscess seen within heart shadow.

large abscess extends mainly to the right of the spine and its left border is only dimly seen through the heart shadow. In figure 424 the main mass of the abscess is situated behind the heart and only a small segment of its periphery is seen in the right cardio-hepatic angle. In figure 110 the abscess is symmetrically disposed about the spine. The interpretation of these shadows of Pott's abscess can at times be made with

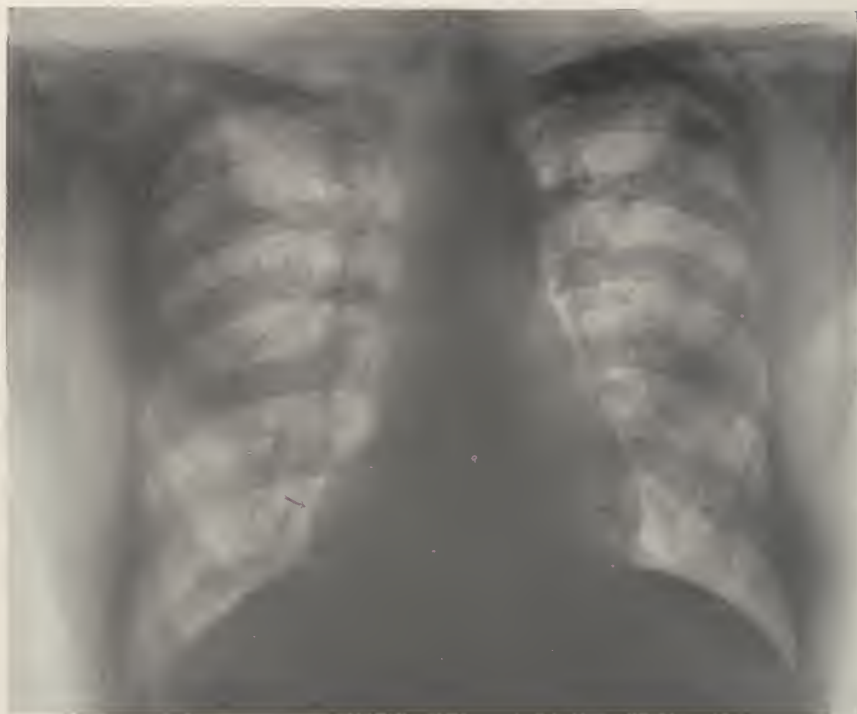


FIGURE 424

Tuberculous abscess of spine almost obscured by the heart. Limits of abscess indicated by arrows.

some confidence from their situation and appearance, especially if the denser shadows of caseous or calcareous material can be recognized within them; as a rule however, the diagnosis will gain its greatest assurance from an examination of the spine and the discovery of tuberculous disease of the vertebrae.

The thoracic deformity resulting from kyphosis is often reflected in a distorted Roentgen picture. The ribs are closer together than normally, obliterating much of the pulmonary fields, so that the recognition of abnormal shadows in the lung becomes impossible. The chest is shortened and the diaphragm is relatively so high that the heart is to a great extent obscured by it and very little is seen above the diaphragm except the aortic arch. (Figs. 425 and 426.)



FIGURE 425

Kyphosis.



FIGURE 426

Thoracic deformity resulting from kyphosis. Heart is barely visible.

The deformity of kyphoscoliosis on the other hand leads to an asymmetry of the chest, the one pulmonary field being almost obscured by the spine and over-lapping ribs. It is worthy of note that even with extreme kyphoscoliosis, the heart frequently maintains its normal position. (Fig. 427.)

The soft tissues of the thoracic wall are a much less frequent cause of abnormal shadows than are the bones. In fact it is uncommon for an inflammatory process such as a cellulitis or even a tumor of the soft tissues to cast any shadow on the plate at all. Only occasionally



FIGURE 427

Thoracic deformity with kyphoscoliosis. Note nearly normal position of heart.

will a tumor of the breast, as in figure 428, produce an abnormal shadow; its appearance in this case was undoubtedly favored by an absence of fat about it, due to the extreme emaciation of the patient.



FIGURE 428

Shadow over the middle of the right lung produced by a carcinoma of the breast.

A very confusing Roentgen picture may result from a peripleuritis, such as is occasionally secondary to a chronic osteomyelitis of a rib. The slight induration of the subpleural tissues will produce a faint homogeneous shadow which it is almost impossible to distinguish from a pleural thickening.

As other occasional causes of abnormal Roentgen shadows, may be mentioned tuberculous cervical lymph nodes. When they are projected over the apical region, they may be confused with pulmonary tuberculous deposits; error can usually be avoided by noting the presence of similar shadows beyond the pulmonary fields. (Fig. 429.) Enlarged



FIGURE 429

Tuberculous cervical and supraclavicular lymph nodes overlying the lung fields.

axillary nodes, even when not calcareous or caseous, as in Hodgkin's disease, may encroach on the lungs at the apex of the axilla and give rise to confusion. (Fig. 430.) Finally, it must be borne in mind that the nipples at times will appear on the plate as small circular shadows, which are very similar to metastatic new growths. Their invariable presence on both sides will preclude any chance for error in their interpretation.



FIGURE 430

Axillary lymph nodes in Hodgkin's disease, overlying the pulmonary field in right axilla. (Arrow.)

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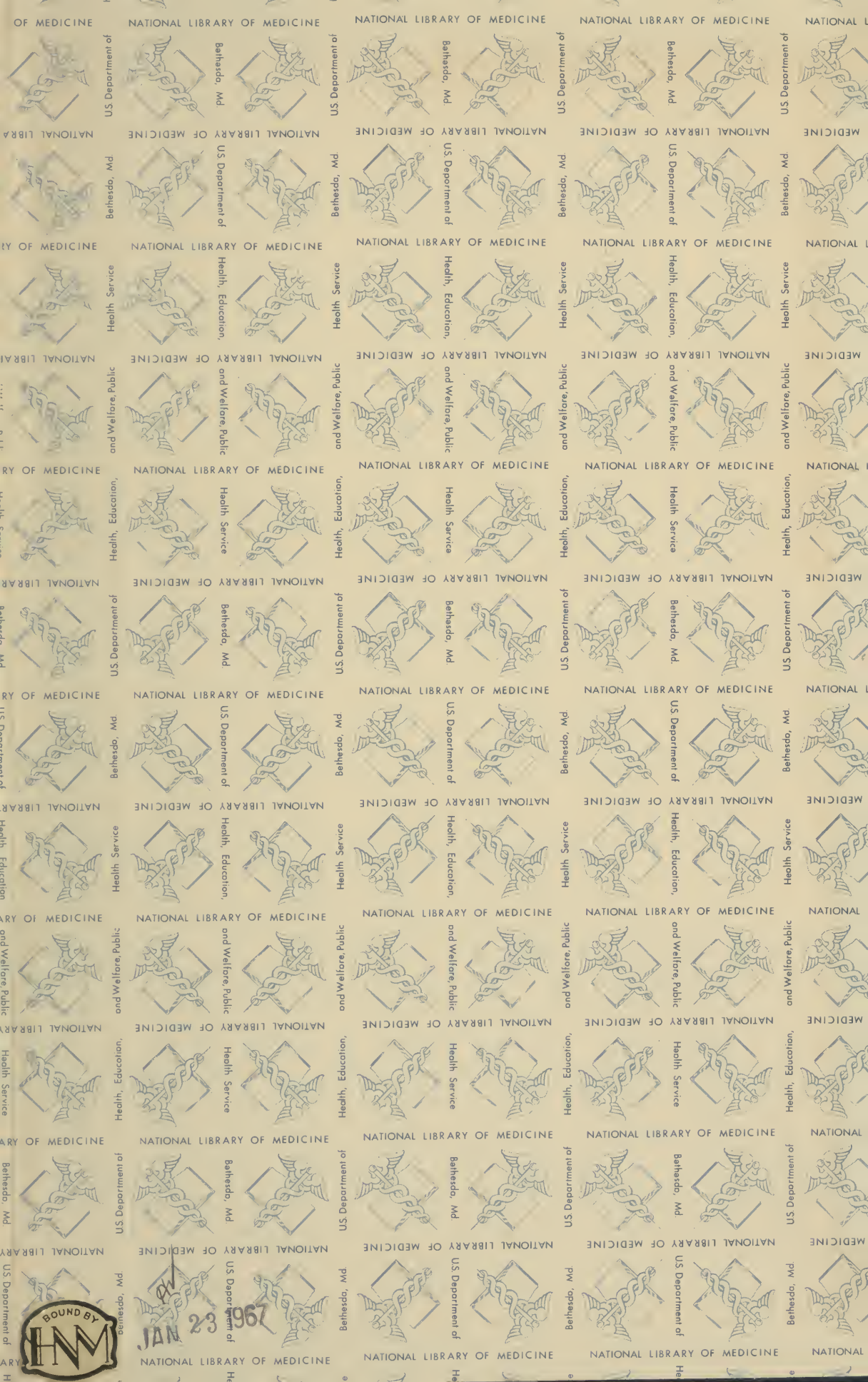
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Unresolved pneumonia,

V

Vertebrae, sarcoma of,
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